Professor Owen
with the author's regards

MR. PAGET'S LECTURES,

DELIVERED AT

THE ROYAL COLLEGE OF SURGEONS,

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LECTURES

ON

INFLAMMATION,

(DELIVERED IN THE THEATRE OF THE ROYAL COLLEGE OF SURGEONS OF ENGLAND,)

BY

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LECTURE I.

Inflammation to be studied as an altered mode of nutrition in a part—Description of the inflammatory state as affecting the several parts chiefly engaged in the process of nutrition—namely, 1st, the Blood-vessels; their enlargement and fulness, producing redness and swelling; their change of shape, and aneurismal dilatations—2nd, the Blood; its mode of movement, especially as observable in the wings of bats, after injury and the application of stimuli; its partial stagnation; its apparent changes; crowding of its red corpuscles; assumed accumulation of its white corpuscles; general effects of the changes of the retarded or stagnant blood—3rd, the Nerves; their state, as indicated by pain, and disturbance of the act of nutrition; transference of this state to other nerves, generating certain forms of sympathetic inflammation—4th. the proper elements of the inflamed part; their influence in determining the phenomena of inflammation; their defective nutrition during the process.

Mr. President and Gentlemen,—In pursuance of the plan which I have followed in former years, of endeavouring to illustrate the successive series of specimens in the Pathological Museum, I come to the subject of Inflammation. Considering the difficulty of the questions to be discussed, I might gladly have avoided them; but the remembrance of the indulgent attention I have received in former years makes me bold to attempt even this subject also; and the more readily, because, certainly, in the whole range of pathological study, none can be compared with it in either interest or importance. It is no more than the truth which Mr. Travers has well expressed in his work on the "Physiology of Inflammation and the Healing Process"—"that a knowledge of the phenomena of inflammation, the laws by which it is governed in its course, and the relations which its several processes bear to each other, is the keystone to medical and surgical science."

I shall not attempt to define inflammation in any set terms; for as yet we are not, I think, in a position to do this. Just definitions cannot be made in any science till some of its broad and very sure principles have been established. Such principles we cannot boast to have yet attained in the study of pathology; and the attempts at precise definitions that have been made hitherto, seem to have led only to confusion, or to false and narrow views of truth. Besides, to define inflammation is the less necessary, because, practically, we all know sufficiently well what the term implies: we know the signs of the presence of the disease in all its chief forms; and, when we watch these signs in any external part, we see them so often followed by peculiar changes in the part, that we are justified in recognizing the changes as effects of inflammation, and in believing that wherever we find them, the similar or corresponding signs of inflammation have preceded them.

But the very difficulty of exactly defining the process of inflammation may be our guide to the most hopeful method of investigating it. When we see such gradual transitions, from the normal process of nutrition to the disease of inflammation, that we cannot draw a definition-line between them, we may be sure that the main laws of physiology are the laws alike of the disease and of the healthy process; that the same forces are engaged in both; and that though interfered with by the conditions of the disease, they are not supplanted or annulled.

Now, such transitions from the normal processes to that of inflammation are not rare. We may trace them, for example, in the gradual passage from the active exercise of the brain, or of the retina, to its "irritation" when overworked, and thence, to its complete inflammation and impairment of structure, after long exposure to what had been a natural stimulus, or to what, in a less degree, might be so. Or, on the introduction of medicines, such as certain diuretics, into the blood, we may trace gradations from the normal increase of the functions of the kidneys, under what is regarded as no morbid stimulus, to their intensest inflammations. Or, again, in the application of an abnormal stimulus, such as that of a heat greater than the natural temperature of the body, where shall we mark the line at which inflammation begins to supervene on health? We may, indeed, say that stagnation of blood, or effusion of liquor sanguinis, shall be the condition *sine qua non* of inflammation; we may call whatever falls short of these, "active congestion," "irritation," or by any other name; but in practice, such distinctions are

often impossible, and sometimes untrue, and in study, the terms are convenient for the sake of brevity rather than of clearness.

Evading, then, the question of the precise definition of inflammation, I shall endeavour, first, to describe the state of an inflamed part, giving to the description such a plan and direction as may best help the chief design of contrasting the inflammatory, with the normal, method of nutrition, and of showing that the immediate causes, and the chief constituents, of the inflammatory state are to be found in alterations of those things which are necessary conditions of the healthy nutrition of a part.

The conditions of the healthy maintenance of any part by nutrition, are—1st, a regular and not far distant supply of blood; 2d, a right state and composition of that blood; 3d (at least in most cases), a certain influence of the nervous force; and 4th, a normal state of the part in which nutrition is to be effected.* All these are usually altered in inflammation.

I. The supply of blood to an inflamed part is altered both by the changes of the blood-vessels, especially by their enlargement, and by the mode in which the blood moves through them.

The enlargement of the blood-vessels is, I suppose, a constant phenomenon in the inflammation of a part; for, although in certain parts, as the cornea, the vitreous humour,† and the articular cartilages, some of the signs or effects of inflammation may be found where there are naturally no blood-vessels, yet I doubt whether these ever occur without enlargement of the vessels of the adjacent parts, and especially of those vessels from which the diseased structure derives its natural supply of nutritive material, and which may therefore be regarded as being its blood-vessels, not less than those of the part in which they lie. Thus, in inflammation of the cornea, the vessels of the sclerotica and conjunctiva are enlarged, and in ulceration of articular cartilages, those of the surrounding synovial membrane or subjacent bone.

The enlargement usually affects alike the arteries, the capillaries, and the veins of the inflamed part; and usually extends to some distance beyond the chief seat or focus of the inflammation. To it we may ascribe the most constant visible sign of inflammation,—the redness, as well as much of the swelling. Its amount is various; it may be hardly perceptible, or it may increase the vessels to twice or three times their natural diameter. Extreme enlargement is admirably shown in Hunter's specimen* of the two ears of a rabbit, of which one was inflamed by thawing it after it had been frozen. "The rabbit was killed when the ear was in the height of inflammation, and, the head being injected, the two ears were removed and dried." A comparison of the ears, or of the drawings from them, shows all the arteries of the inflamed ear three or four times larger than those of the healthy one, and many arteries that in the healthy state are not visible, are, in the inflamed state, brought clearly into view by being filled with blood.

I have repeatedly seen similar enlargements of both arteries, and veins, and capillaries in the stimulated wings and ears of bats. The like phenomena occur in the webs of frogs, and other cold-blooded animals; but in these, I think, the amount of enlargement is generally less.†

The redness of an inflamed part always appears more than is proportionate to the enlargement of its blood-vessels; chiefly, because the red corpuscles are much more closely crowded than they naturally are in the blood-vessels. The vessels of an inflamed part are not only dilated, but appear crammed with the red corpuscles, which often lie or move almost as if no fluid intervened between them: their quantity appears increased in far greater proportion than that of the liquid part of the blood.

This peculiarity is even more manifest in the frog than in the bat; for in the former, the crowding of corpuscles may occur in vessels that appear to have undergone no change of size on the application of the stimulus.‡

Another, but a minor, cause of the increased redness of the inflamed part is sometimes to be observed in the oozing of the colouring matter of the blood-corpuscles, both into all the interspaces between them, and through the walls of the small vessels into the adjacent tissue. During life this may be noticed, especially

* See Lectures on Nutrition, &c., in the MEDICAL GAZETTE, 1847.

† See, especially, a case by Mr. Bowman, in his "Lectures on the Eye," p. 124.

* Pathological Museum of the College, No. 71. Catalogue, vol. i. p. 33. See also Hunter's Works, Vol. iii. p. 322, and Pl. xx.

† Emmert, who is among the few that have measured it, says it is equal to one-half or one-third of the normal diameter of the vessels. Bidder denies it altogether. See Henle's and Pfeufer's Zeitschrift, B. ii. and iv.

‡ I do not more particularly refer to what is described as the encroachment of the red blood-corpuscles on the lymph-spaces, or the layer of fluid that lies in apparent rest adhering to the walls of the vessels. The too-pointed description of this layer has led to very exaggerated notions concerning it: its existence is certain, but it is too thin for any blood-corpuscle to lie at rest in; and when white corpuscles remain by the walls of the vessels, it is evident that they do so more because of their own adhesiveness than because a small portion of the fluid about them is at rest.

when the blood is stagnant in the vessels, and it may give them a hazy, ruddy outline, but it is generally much more considerable after death, when we may ascribe to it no small portion of the redness that an inflamed part may still present.

In the state of inflammation no new blood-vessels are formed. Many more may come into view than were at first seen in the part; but these are only such as were invisible till the flood of blood-corpuscles filled and distended them. So it was in the rabbit's ears; in the healthy ear no trace can be seen, with the naked eye, of any vessels corresponding with one of the largest, or with many of those of inferior size, in the inflamed ear. So it is, too, in microscopic examinations. Within half an hour after stimulating a bat's wing, many vessels may come into view which could not be seen before, and with which none can be seen corresponding in the other wing, though doubtless such vessels exist there of smaller size.

It is only when the inflammation has subsided, and the lymph exuded from the blood-vessels begins to be more highly organised, that new vessels are formed, and pass into the lymph, as if for the maintenance of its increase or development.* So long as the inflammation lasts, the intensest redness in parts naturally colourless,—even such as we see in acute inflammation of the conjunctiva, or yet more remarkably in those of periosteum,† or in congestion of the stomach,—is due to the enlargement of the natural blood-vessels, to their admitting crowded red corpuscles, and in a much less degree, and, perhaps, in only certain cases, to the diffusion of the colouring matter of the blood.

With the enlargement of the blood-vessels a change of shape is commonly associated. Being usually elongated as well as dilated, they are thrown into curves and made more or less wavy or tortuous. Thus we may see the larger vessels in an inflamed conjunctiva,—or, more plainly, the subperitoneal arteries in cases of peritonitis; so, too, they are represented in the inflamed rabbit's ear.‡

A more remarkable change of shape of the small vessels of inflamed parts is that in which they become aneurismal or varicose. The first observations of this state were published, I believe, by Kölliker and Hasse, in an account of a case of inflammatory red softening of the brain, in which many of what, at first sight, appeared to be points of extravasated blood, proved to be dilatations of capillary vessels filled with blood. After this they found the same changes, but in a much less degree, in some cases of inflammation artificially excited in the brains of rabbits and pigeons.* Many, as well as myself, have since made similar observations, most of which, however, seem to show that the peculiar dilatation has its seat in the small arteries, not in the capillaries of the inflamed part. The diagrams illustrate some of the dilatations observed in vessels of the brain by Kölliker and Hasse; and of those seen by Bruch† in the peritoneum of a dog after a wound in the abdomen; one from a specimen of diseased ovary, described by Professor Harting, and given to me by Dr. Van Leeuwen; and some from a case of inflammation, or extreme congestion of vessels, in a fringe of false membrane on a pericardium. The several figures represent various forms and amounts of partial dilatation. Some are like gradual fusiform dilatations of the whole circumference of the vessel; some like shorter and nearly spherical dilatations of it; some like round, or oval, or elongated pouches, dilated from one side of the wall: in short, all the varieties of form which we have long recognised in the aneurisms and aneurismal dilatations of the great arteries may be found in miniature in the small vessels of such inflamed parts.

Frequently, however, as this state of the small vessels has been observed (and I believe some measure of it may be found in the inflammations of most membranes), yet, I think, we may not assume it to have a necessary or important connection with the other phenomena of inflammation. It may be as a mere accident to the inflammatory process, and due to some weakening of the vessels, which renders them unable to resist uniformly the increased pressure of the blood; or, perhaps, in some cases, as Mr. Quekett has suggested to me, the pouch-like dilatations may represent a disturbed effort for the production of new blood-vessels by dilatation, or out-growth of the walls of those already extant.

Such is the ordinary state of the blood-vessels of an inflamed part: all dilated and elongated, tensely filled with blood, of which the red corpuscles are in excess,

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* Mr Hunter held this opinion; but more lately the contrary one has been commonly held. See his Works, vol. iii. p. 322.

† As illustrated in Mr. Stanley's plates, plate vii. fig. 1, which represents a specimen in the Museum of St. Bartholomew's, Series i. No. 195: the whole inner surface of the inflamed periosteum of a tibia is bright scarlet.

‡ Sometimes such tortuosity makes the vessels appear varicose, as in a case by Reichert, in Müller's Archiv, 1847.

* See, regarding all the cases, Kölliker's paper in his Zeitschr. für wissensch. Zoologie, B. i. S. 262, et seq.

† Henle and Pfeufer's Zeitschrift, Bd. v. S. 65, and Taf. i.

often wavy and tortuous, and sometimes variously aneurismal.

But the supply of blood to an inflamed part is affected by its mode of movement, as well as by the size of the blood-vessels; this, therefore, I must now describe.

Nearly all the observations hitherto recorded on the morbid changes in the movement of the blood have been made with the webs of frogs; and it has been objected that it is not safe to apply conclusions drawn from them to the case of warm-blooded animals. I have therefore employed in my recent observations the wings of bats, in which (when one has acquired some art in quieting them with chloroform or gentle management) nearly all the phenomena of the circulation, as affected by the application of stimuli, may be watched as deliberately as in the frog, and in some respects even more clearly.

I think we may believe that what may be seen in the wings of bats occurs, in the like circumstances, in all warm-blooded animals. It is true that, like the other hybernants, the bats, while they are in their winter-sleep, resemble the cold-blooded animals, in that their temperature is conformed to that of the external air, and scarcely exceeds it. It is true, also, that when they are ill nourished, their temperature, even in their active state, is comparatively low, ranging from 65° to 80° F., in an atmosphere of 60°; and that generally they are liable to much greater diversities of temperature than our own bodies are.* Yet, since in the development of their nervous system, and the commensurate development of their heart and respiratory organs, and in the close reciprocal relations in which these act, the bats resemble the other warm-blooded vertebrata, we may, I think, fairly assume a close resemblance also in their processes and conditions of nutrition; especially we may assume that this resemblance exists while they are in their active period of life, and in good health, as those were in which nearly all my observations were made.

Before describing the effects of irritation, it may be well to point out a peculiarity in the arrangement of the blood-vessels of the bat's wing. The principal arteries and veins lie side by side at each border of the metacarpal bones and phalanges, and the intervening membrane contains their numerous branches and capillaries. But, very generally, the arteries of the second and third order of branches pass into veins of corresponding size, without any intermedium of capillaries. The capillaries are rather in the position of off-sets from the continuous channels of arterial and venous loops, than in their more ordinary relation as intermediate canals, leading from arteries to veins. I know not to what this condition may have reference, or what purpose it may serve; but, in relation to the phenomena of inflammation, it appears to have no other effect than that, through the abundant anastomosis, a great obstacle to the movement of the blood is scarcely likely to occur.

The simplest effects upon the blood-vessels are produced by a slight mechanical stimulus. If, as one is watching the movement of blood in a companion artery and vein, the point of a fine needle be drawn across them three or four times, without apparently injuring them or the membrane over them, they will both presently gradually contract and close. Then, after holding themselves in the contracted state for a few minutes, they will begin again to open, and gradually dilating, will acquire a larger size than they had before the stimulus was applied.

Simple as this observation is, it involves some cardinal facts in our pathology. It illustrates, first, the contractile power of both arteries and veins; and, by the manner of their contraction, which follows at some interval after the application of the stimulus, and is slowly accomplished, it shows that their power of contraction is like that of parts with simple or organic muscular fibres. And one may notice here the illustration of the stoppage of hæmorrhage from small vessels. In one cut we may divide a hundred such vessels as these on the surface of a stump, and they may cease to bleed in a few seconds: doubtless, the very stimulus of the knife, while dividing them, has made their walls contract and close.

* For instance, I found the temperature of a strong and active Noctule-Bat (*Vespertilio Noctula*) thus various in two days:—

April 29th, at noon, after he had been nearly two hours under the influence of chloroform, and on awaking had been struggling very actively, his temperature was 99° F. At 9 P.M., having meantime been quiet, hanging by his hind feet, and looking sickly, his temperature was only 70°. When disturbed he became very fierce and active, shrieking and biting the bars of his cage; and at 9h. 40m. his temperature was 92°. Soon after this he became quiet again, and at 10h. 30m. his temperature was 80°. The temperature of the atmosphere during these examinations had gradually increased from 61° to 67°.—April 30th, at 8 A.M., he was feeble, but not torpid: the temperature of the room during the night had been between 40° and 45°, and was now 57°; the temperature of the bat was only 59°. At 11 A.M., after struggling violently for half an hour, it rose to 69°. After being long under chloroform, and nearly dying, he remained all the afternoon only one or two degrees warmer than the atmosphere. But at night, at 12h. 15m., he recovered and became active, and, while the atmosphere was at 65°, he was at 85°. At 12h. 40m., after being made very fierce, he was at 88°; and at 1h. 30m. remained at 85°. Next morning he was again scarcely warmer than the atmosphere. The temperature was always taken with a small thermometer applied to the surface of the abdomen.

But, again, the experiment shows the vessels reopening and becoming wider than they were before, either yielding more to the pressure of the blood which previously they resisted with more strength, or else dilating, as of their own force, with that which Mr. Hunter called active dilatation, and compared with the act of dilatation of the os uteri. In whichever way the dilatation is effected, whether it be active or passive, the vessels will not at once contract again under the same stimulus as before affected them. The needle may be now drawn across them much oftener and more forcibly, but no contraction ensues, or only a trivial one, which is quickly succeeded by dilatation. Yet with a stronger stimulus, such as that of great heat, they will again contract and close. And such a contraction excited by a cautery may last more than a day, before the vessels again open and permit the flow of the blood through them. So that in this we have an illustration of the secondary hæmorrhages from vessels which, after their first closure, have not been sealed by the coagulating blood, or the exudation of lymph,—as well as an illustration of the effect of the cautery or of hot water in again checking such hæmorrhages, and more permanently closing up the vessels.*

Moreover, we may observe in this experiment the adapted movement of the blood. As the vessels are contracting the blood flows in them more slowly, or begins to oscillate; nay, sometimes, even before the vessels begin visibly to contract, one may observe that the blood moves more slowly in them, as if this were the first effect of the stimulus: nor am I sure that I have ever seen (what is commonly described) the acceleration of the flow of blood in the contracting vessels. Such an acceleration, however, is manifest, as the vessels re-open; and as they dilate, so, apparently in the same proportion, does the flow of blood through them become more free, till at length it is quite manifest that they are traversed by both fuller and more rapid streams than passed through them before the stimulus was applied. How long this state may last depends on many circumstances hard to estimate: but at length it ceases, and the vessels, and the circulation through them, assume again their average or normal state.

Such are the effects of the simplest stimulus of blood-vessels. Corresponding observations have been made on those of frogs, especially by E. H. Weber;* but he used the electro-magnetic stimulus: and from the relation of his experiments we may, I think, conclude that the vessels of the warm-blooded animals may be affected by much less severity of stimulus than those of cold-blooded ones. Moreover, the contraction of the veins in his experiments was very slight, and sometimes not discernible; so it always appears to be in frogs; but in bats it is quite as well marked as that of the arteries.

The effects of the application of other stimuli to the wings of bats correspond in kind with those I have just described, but differ in degree and extent. If a drop of acetic acid, or of tincture of capsicum, of turpentine, or of ethereal solution of cantharides, be placed on a portion of the wing, or washed over it, one sees a quickly ensuing dilatation of the bloodvessels, and a rapid flow of blood through them all. I am not sure that the dilatation is preceded by contraction. Certainly the contraction is very slight, if it occurs at all; but the dilatation is usually much more extensive. When the stimulus has been applied to only one small spot upon the wing, the whole of the bloodvessels in the corresponding metacarpal space, and even those of the adjacent spaces, may enlarge. One might imagine that the dilatation of vessels was due to an increased action of the heart, if it were not that (as I think) it is always greater at the very point to which the stimulus was applied than in any other part of the same wing, and is never at all imitated in the corresponding parts of the opposite wing.

As I have already said, the streams of blood are more rapid in the dilated vessels than in the others, and than in those of the opposite wing. They are also more steady; for in what appears to be the natural state of the circulation in the bat's wing, it is not unfrequent to see an occasional oscillation in the venous stream, an occasional stoppage, or back-current, and then a more forcible rush forward; but these are rarely seen when the stimulus has produced the effect that I have described.

The state which is thus induced by stimuli is what is commonly understood by the expressions "active congestion," or "determination of blood," in a part. It consists, briefly, in general enlargement of the bloodvessels of the part, with an increased velocity of the blood in them. It is, probably, just such a state as this that is felt by suckling women in what they term the "flow of milk," which is no doubt an increased flow of blood into the mammary gland just before

* For the control of hæmorrhages something more than the contraction of the vessels is required, that their orifices may be *sealed* before they can again dilate; and it is probably to the absence of this something, more than to any condition of the vessels, that we must look for the explanation of secondary hæmorrhages from small vessels.

* Müller's Archiv, 1847.

a quicker secretion of the milk. Less normally, it is such a state as this that we observe in the skin after the application of mustard, or sharp friction, or a heat from 20° to 50° above its own, or in the conjunctiva when stimulated by dust that is soon dislodged; and such we may believe to be the condition of many internal organs when we might doubt whether they are inflamed, or are only very actively discharging their natural functions. Herein, indeed, in what I have described, is one of the pieces of neutral ground between health and disease: a step in one direction may effect the return to health, in another the transit to what all might admit to be the disease of inflammation.

Now this transit appears to be made when the circulation, which was rapid, begins to grow slower, without any diminution, but it may be with an increase, of the size of the vessels. This change one may see in the bat's wing. After the application of such stimuli as I have already mentioned, the movement of the blood may become gradually slower, till, in some vessels, it is completely stagnant. But a corresponding state is better seen after such an injury as that of a fine red-hot needle driven into the membrane of the wing, or through it.

The first effect of such an injury (in addition to the charring and searing of the membrane, the obliteration of its blood-vessels, and the puckering of the portion of it adjacent to the burn) is to produce contraction of the immediately adjacent arteries and veins. They may remain closed, or, as I have already described, after being long closed, may again open, and become wider than they were before. This dilatation follows more certainly, and perhaps without any previous contraction, in the arteries and veins at a little distance from the burn. In these there speedily ensues such a state of "determination of blood" as I have already described: in arteries and veins alike the stream is full and rapid; and the greater accumulation, as well as the closer crowding of the red corpuscles, makes the vessels appear very deep coloured. The contrast of two diagrams, showing the natural and the stimulated conditions, illustrates this difference sufficiently well. The vessels of the one, nearly twice as large as those of the other, darker, and more turgid with blood; and, in the one, numerous capillaries which are not visible in the other. But diagrams cannot show the changes in the mode of movement. Close by the burn, the blood which has been flowing rapidly begins to move more slowly, or with an uncertain stream,—stopping, or sometimes ebbing, and then again flowing on, but, on the whole, becoming gradually slower. Thus it may, at length, become completely stagnant; and then, in the vessels in which it is at rest, it seems to diffuse and change its colour, so that its crowded corpuscles give the vessels a brilliant carmine appearance, by which, just as well as by the stillness of the blood, they may always be distinguished. As one surveys an area surrounding this part in which the blood is stagnant, or moves slowly, one sees the streams increasing gradually in rapidity. And often, when there is stagnation in a considerable artery, one may see the blood above or behind it pulsating with every action of the heart, driven up to the seat of stagnation, and thence carried off by the collateral branches; while in the corresponding vein it may oscillate less regularly, delaying till an accumulated force propels it forward, and, as it were, flushes the channel.

Again, in the same area as that in which the movement is pulsatile or oscillating, and in the area beyond it, one sees the full and rapid and more numerous streams of "active congestion;" and these may extend over a space altogether uncertain.

Such is the general condition of the circulation round a part thus inflamed; but the varieties in lesser points that may be presented cannot be described. These must be seen; and, indeed, the whole sight should be viewed by every one who would have in his mind's eye a distinct image of what in practice he must often too obscurely contemplate.

The phenomena that I have described as seen in the bat's wing correspond very closely with those observed in the frog's web. Only, I think, the stagnation of blood is neither so constant nor so extensive in the bat: it is seen in portions of single vessels, rather than in districts of vessels,—usually in corresponding portions of arteries and veins, as they lie side by side. The stagnation usually extends into such branches as may be given off from the vessels that are its principal seats; and three or four such seats of stagnation may appear placed irregularly about the burn, or other focus of the inflammation; but I have never seen a general stagnation of blood in all the vessels of even a severely stimulated part. My impression is that in strong and active warm-blooded animals stagnation of blood would be found in only the most severely inflamed parts: in others, I think, retardation alone would exist.

To sum up now what concerns the supply of blood in an inflamed part. We seem to have sufficient evidence that, in general, in the focus of the inflammation, blood is present in very large quantity, distending all the vessels, gorging them especially

with red corpuscles, but often moving through them slowly, or even being in some of them quite stagnant; that all around this focus, the vessels are as full, or nearly as full, as they are in it, but the blood moves in them with a quicker stream, or may pulsate in the arteries, and oscillate in the veins; that, yet further from the focus, the blood moves rapidly through full but less turgid vessels; and that this rapidity and fulness are not to be ascribed merely to the blood, which should have gone through the inflamed part, being driven through collateral channels, but is such a state as is commonly understood as an "active congestion," or "determination of blood," in the part.

I have already said that we may believe that what is seen in the bat represents fairly the state of inflamed parts in all warm-blooded animals. I am quite conscious that the most one can see with the microscope is but a faint picture of such inflammations as we have to consider in practice; that it is very trivial in both its appearance and its results. Still, it is a picture of a disease of the same kind; and a miniature, even faintly drawn, may be a true likeness. Besides, all that can be observed of the complete process of inflammation in man is consistent with what we can see in these lower and lesser creatures. The bright redness of an inflamed part testifies to the fulness of its bloodvessels, and the crowding of the red corpuscles; the occasional duskiness or lividity of the focus is characteristic of stagnation; the throbbing in the part, and about it, and the full hard pulse in the ministrant arteries, are sure signs of obstruction to the passage of blood; the gush of blood on cutting into the tissues near an inflamed part, or in bleeding from one of their veins, tells of the determination of blood in these, and of the tension in which all the containing bloodvessels are held.

It is particularly to be observed that the stagnant or retarded blood is not apt to coagulate. I have found it fluid after at least three days' complete stagnation, and so I believe it would remain till it is cleared away, unless the part sloughs. In the latter case it would coagulate, as it does in carbuncles and the like, which hardly bleed when we cut them through; but, so long as the blood is fluid, though stagnant, it may be driven from the vessels with full force as soon as an easy exit for it is made by cutting into the inflamed part, or opening one of its large veins. I need here only refer to Mr. Lawrence's well-known and instructive experiment. In a patient with an inflamed hand he made similar openings into veins in both arms. From the vein on the diseased side, three times more blood flowed than from the vein in the healthy arm, in the same time; and this increased flow represented at once the greater determination of blood about the focus of the inflammation, and the greater tension in which were held the walls of the blood vessels, and, indeed, all the integuments of the inflamed and swollen part.

Now, to what can we ascribe these changes in the movement of the blood? In all the pathology of inflammation no problem seems more difficult than this principal one.

It has been commonly said that, as the vessels contract, therefore the movement of blood becomes more rapid in them, as when a river entering a narrow course moves through it with a faster stream; and that then, as the vessels widen, so the stream becomes, in the same proportion, slower. But this is far from true. The stream often becomes slower as the artery or vein becomes narrower by contraction; and then, as the tube again dilates, the stream grows faster; and then, without any appreciable change of size, it may become slower again, till complete stagnation ensues in at least some part of the bloodvessel. I think I can be quite sure that the velocity of the stream in any vessel of an inflamed part is not determined by the diminution or enlargement of the channel. Without change of size, the stream may be seen decreasing from extreme velocity to complete stagnation.

On what the alteration of movement of the blood in such a case depends I cannot tell; but we have facts enough to justify such an hypothesis as that there may be some mutual relation between the blood and its vessels, or the parts around them, which, being natural, permits the most easy transit of the blood, but, being disturbed, increases the hindrances to its passage. Such hindrances appear to be produced by the addition of salts of baryta, or of potash, to the blood: the presence of an excess of urea in the blood probably produces the like effect: and such facts as these make the hypothesis I have referred to not unreasonable. At any rate, the belief that the more or less rapidity of movement of blood through small vessels may depend on other than evident mechanical relations, cannot appear absurd to any one who has seen the movements of fluid in the Chara, or Vallisneria, or any such plants, in which a circulation is maintained without any visible source of mechanical power.

II. I mentioned, as the second condition necessary to the healthy nutrition of a part, a right state and composition of the blood.

In a former course of lectures* I pointed out that, by this state, we must understand not merely such purity of the blood that chemistry cannot detect a wrong constituent in it, or a wrong quantity of any of the normal ones, but that natural constitution of the blood by which it is exactly adapted to every tissue that it has to nourish,—with an adaptation so exact that chemistry cannot approach to the determination of whether it is maintained or lost.

That this adaptation is disturbed in many cases of inflammation is proved by the instances in which inflammations plainly have their origin in morbid conditions of the blood. But I fear that the nature of this disturbance cannot yet be chemically expressed, and that the facts which chemistry has discerned in the condition of the blood in inflammations cannot yet be safely applied in explanation of the local process. For, first, we observe the phenomena of inflammation where we cannot suppose the whole blood disordered,—as after the application of a minute local stimulus, such as a foreign body on the conjunctiva; and, secondly, among the changes observed in inflammatory blood, the principal one—namely, the supposed increase of fibrine—is ambiguous: it may be at once an increase of fibrine and of the white corpuscles of the blood. These two constituents of the blood, the fibrine and the white or rudimental corpuscles, cannot be well separated by any process yet invented; and in all the estimates of fibrine, whether in health or in disease, the weight of the white corpuscles is included. Now, in many inflammations, these corpuscles are increased, and we have no means of clearly ascertaining how much of an apparent increase of fibrine is really such, and how much is due to the corpuscles entangled in the fibrine. Till this can be settled, I think we may not deduce any of the local phenomena of inflammation from the increase of fibrine in the blood; neither, more assuredly, can we trace, as some do, the fever and other general signs of inflammation to the abstraction of fibrine and albumen by the exudation from the blood.†

The other changes of the blood in inflammation—the diminution of its red corpuscles and increase of water—are even less adapted to explain any of the phenomena of the local process. Whatever may be their strength or value as facts, they are as yet isolated facts, such as one cannot weave into the pathology of the disease.

I fear, too, that the structural condition of the blood will not, more than the chemical, help us to explain the phenomena of inflammation. Some of our most worthily distinguished physiologists have ascribed much to the existence of large numbers of the white blood-corpuscles, and their accumulation in the vessels of the inflamed part: indeed, they have taken this for the foundation of nearly their whole doctrine of inflammation, ascribing to it both the stagnation of the blood and the changes it is presumed to undergo,—such as the increase of the fibrine, and many others. But the facts on which they have rested are unsound: their observations have been made on frogs, and do not admit of application to our own case, or, perhaps, to that of any warm-blooded animal. In many frogs, especially in those that are young, or sickly, or ill-fed, the white corpuscles are abundant in the blood: they are rudimental blood-cells, such as may have been formed in the lymph or chyle; and in these cases they are either increasing quickly in adaptation to quick growth, or else increasing because, through disease or defective nutriment, although their production is not hindered, yet their development into the perfect red blood-cells cannot take place. In either case, their peculiar adhesiveness making them apt to stick to the walls of the blood-vessels, they may accumulate in a part in which the vessels are injured or the circulation is slow, and thus they may sometimes augment the hindrances to the free movement of the blood. But I believe nothing of the kind happens in older or more healthy frogs, or in any ordinary inflammation in the warm-blooded animals. I have drawn blood from the vessels in the inflamed bat's wing, in which it was quite stagnant, and have found not more than one white corpuscle to 5000 red ones. I have often examined the human blood in the vessels of inflamed parts after death, and have found no more white corpuscles in them than in those of other parts. In blood drawn from inflamed parts during life, I have found only the same proportion of white corpuscles in them as in the healthy parts of the same person. I therefore cannot but accord with the opinion often expressed by Mr. Wharton Jones and Dr. Hughes Bennett, that an especial abundance of white corpuscles, *i. e.* of rudimental blood-cells, in the vessels of an inflamed part, is neither a constant nor even a frequent occurrence; and I believe that, when such corpuscles are nu-

* On Nutrition, &c., published in the Medical Gazette for 1847.

† The whole of this part of the current pathology of inflammation seems to have been too hastily constructed. The local changes observed in the frog have been used to explain the chemical changes of the blood in man, although no such changes have been proved in the blood of the frog; and the changes in the blood of man have been used to explain in him the existence of local phenomena which are assumed, but have never been proved, to be similar to those observed in the frog.

merous in an inflamed part, it is only when they are abundant in the whole mass of the blood. Now, as already stated, they are thus abundant in some cases of inflammation, especially, I think, in those occurring in people that are in weak health, and in the tuberculous; but, even in these cases, I have never seen an instance in which they were present in nearly sufficient quantity to add materially to the obstruction of the blood in the inflamed part, nor one in which any influence of theirs could be suspected to alter peculiarly the constitution of the blood therein.

Mr. Wharton Jones was the first to describe accurately a remarkable condition presented by the red blood-cells in inflammations. As soon as ever a drop of inflammatory blood is spread out thinly, the corpuscles seem to run together and adhere in long rows or clusters, and these holding together give the clot thus formed on a slip of glass the peculiar mottled pink and white appearance which Mr. Hunter observed as one of the characters of inflammatory blood. The same condition is observed in the blood of pregnant women, and appears natural in that of horses; and in all these cases it may be regarded as the chief cause of the formation of the buffy coat, inasmuch as the clustered blood-cells, sinking rapidly, will generally subside to some distance below the surface of the liquid part of the blood, before the coagulation of the fibrine is begun.

Some have supposed that a similar adhesion of the blood-cells may occur in the vessels of an inflamed part. I have seen nothing of the kind in either the inflamed bat's wing or in the vessels of inflamed organs examined after death. When the blood is not stagnant, the corpuscles are indeed closely crowded, but they are not clustered, nor do they appear adherent: neither does such clustering appear even in stagnant blood; the change here appears to be a diffusion of the colouring matter, so that the outlines of individual blood-cells cannot be seen, and all the contents of the vessel present an uniform bright carmine tint.

But although we can see so little of the changes that may ensue in blood thus stagnant or much retarded, yet we may be nearly sure that the blood in an inflamed part does undergo important changes, when we remember what general effects, what constitutional disturbance, may ensue in the train of an inflammation of purely local origin. Changes probably ensue in the blood similar to some of those that we shall have to trace in the lymph effused from it into the parts around the vessels; possibly particles of fibrine may coagulate in it, and corpuscles of lymph or pus may be formed and degenerate within it; and these, when the stagnation is not constant, or is incomplete, or is passed away, may be carried into the general circulation, infecting the whole blood, exciting general disturbance, as in traumatic fever, or producing various and wide-extended suppurations, as in the purulent diathesis following local injury. All these, and many other concomitants of inflammation, may be reasonably ascribed, at least in part, to the changes that the blood undergoes in the inflamed tissue; but I must repeat that nothing that either the microscope or chemistry has yet discerned will suffice to explain these changes: they belong rather to the theory than to the facts of inflammation.

III. The third enumerated condition for the healthy nutrition of a part is a certain influence of the nervous force. The change that this undergoes in the inflammatory mode of nutrition is, therefore, next to be considered.

That in the higher vertebrata some nervous force is habitually exercised in the nutrition of all the parts in or near which nerves are distributed; and that it is exercised, not merely in affecting or governing the size of the blood-vessels of the part, but, with a more direct agency, as being one of the forces that concur in the performance of the plastic act;—these things may, I believe, be amply proved; and I shall again have occasion to refer to them. But as we have no exact knowledge of the nervous force, or of the manner in which it operates in natural formation, so neither can we tell how its operation is affected while it shares in the production or maintenance of inflammation.

The expression that the nerves of an inflamed part are in an "excited" state, is suggested by the existence of pain,—by a slight stimulus being acutely felt,—by the natural heat, or a slight increase of the heat, being felt as a burning,—and by the part being, even independent of any known stimulus, the seat or source of subjective pains and heat. But the very frequent cases in which pain exists, and abides long, without any other sign of inflammation, and the cases in which the pain bears no kind of proportion to those other signs, or to the effects, of inflammation,—these may suggest that, besides this "excited" state of the nervous force which is felt as pain in the inflamed part, there may be some other state by which the nervous force is more intimately connected with the inflammatory process,—a state of disturbance, which may, indeed, be felt as pain, but

which more properly affects the influence of the nervous force in the process of nutrition.

We obtain some evidence of the existence of such a state when we observe that, without relation to pain, it is communicable from the nerves of inflamed parts to those of other parts; in which parts, then, a kind of sympathetic inflammation may be generated. This transference or communication of the disturbance of nervous force is, indeed, evident enough in relation to that state which is felt as pain; for pain is not limited to the inflamed part, but is diffused around it, and is in sympathy often felt where no other sign of inflammation exists. But besides, and sometimes, I repeat, independent of this condition which is felt as pain, the inflammatory condition of the nervous force may be similarly communicated or transferred. The simplest may be the most proving instances. Whoever has worked much with microscopes may have been conscious of some amount of inflammation of the conjunctiva in consequence of over-work. Now the stimulus exciting this inflammation has been directly applied to the retina alone; and I have often had a slightly inflamed left conjunctiva, after long working with the right eye, while the left eye has been all the time closed. I know not how such an inflammation of the conjunctiva can be explained, except on the supposition that the excited state of the optic nerve is transferred or communicated to the filaments of the nerves of the conjunctiva, generating in them such a state as interferes with its nutrition. It is true that in these simpler cases the retina is not itself evidently inflamed; but after yet severer stimulus it commonly is so, and in these the conjunctiva shares in the evil effects of the communicated stimulus,—effects which we cannot ascribe to any alteration in the blood, or the size of the blood-vessels.

I may mention another case: the occurrence of inflammation of the testicle in cases of severe irritation of the urethra. The most unexceptionable cases of the kind are those in which the irritation is produced by a calculus impacted in a healthy urethra. I have here a specimen* in which extensive deposits of lymph and pus are seen in the testicle of a man in whose urethra a portion of calculus was impacted after lithotrity. Here is such an inflammation as we cannot refer to disease of the blood, and attended by such changes as we cannot explain by any enlargement or paralysis of the blood-vessels: nor do I know how it can be at all explained except by the disturbance of the exercise of the nervous force in the testicle, which disturbance is excited by transference from the morbidly affected nerves of the primary seat of irritation.

In like manner, I believe that the extension or transference of inflammation after pain may be ascribed, at least in part, to the coincident transference of the disturbed plasturgic force of the nervous system. In paroxysms of neuralgia, we see sometimes a transient inflammatory redness or œdema of the part; so, when a more abiding pain has been excited, by sympathy with some inflamed part, there may presently supervene the more palpable effects of inflammation.

I feel that, in discussing such a point as this, one passes from the ground of demonstrable facts; but there is less fault in this than in the belief that the very little we can see of a morbid process can guide us to its whole pathology. When we look at an inflamed part, we should not think that, if we could see its blood-vessels and test its blood, we should detect all that is in error there: rather we should think that all the forces are at fault which should be concurring to the due maintenance of that part; and while we are ignorant of the nature of some of these forces, it is better that their places in our minds should be occupied by reasonable hypotheses, than that they should be left blank, or be overspread with the tinge of one exaggerated theory, such as that which ascribes all inflammation to a change in the state of the small blood-vessels.

IV. The last condition necessary to healthy nutrition in a part is the natural or healthy state of the part itself.

How, in the inflammation of a part, its proper elements are altered, we cannot say, for in the early stages of the process we can rarely see a change in them; nearly the whole of the visible error is in the blood-vessels and their contents, and in the interspaces between the proper elements of the part. Hence it is not surprising that many good pathologists have held such opinions as that the blood-vessels are the mainsprings of the process of inflammation, and that it essentially consists in an altered relation or reciprocal action between the vessels and the blood.

Yet it seems more reasonable to think that the morbid process may be determined by the state of the proper elements of the part—of its cells or filaments, or, more probably, of the material in it which is in progress of development. For, for example, some of the phenomena of inflammation

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* From the Museum of St. Bartholomew's Hospital, Ser. 28, No. 55.

may ensue, through injury or disease, in parts that have neither nerves nor blood-vessels. We have instances of this in the deposits of lymph, or the other consequences of severe injuries, in the cornea and vitreous humour,—parts which, if ever they are vascular, become so only after the effusion of lymph in them. Such, too, are seen in the ulcerations of articular cartilage, in which the vascular phenomena of inflammation are confined to the adjacent tissues. We cannot in these cases ascribe the inflammation to an alteration of the relation between the blood and blood-vessels, for blood-vessels do not exist in the part in which the inflammation has its seat.

Further evidence that some morbid state of the proper elements of a part, or their altered relation to the blood, may determine both the advent and some of the results of inflammation, is afforded by analogy. In the natural state, it is almost always noticeable that the condition of each part determines the amount of blood to be supplied to it, and in some measure, also, the rate of movement and the mode of disposal of the blood. Thus, in the embryo, each part is formed in rudiment before it receives any blood, and the increasing supply of blood never precedes, but follows, and is adjusted to, the increasing development and growth. In later life there are, indeed, some cases in which an accidentally increased supply of blood is followed by an increased growth of parts, as in some hypertrophies and morbid growths, or as in parts transplanted from less to more vascular structures; but the more usual and normal course is that the increasing supply of blood follows, as a consequence, the increasing growth.

We might, therefore, well expect that an alteration in the state of a part itself,—*i. e.*, not of its blood-vessels or its nerves, but of its proper elements, or, more especially, of those which are in progress of development,—would be a constant concomitant of the other changes that make up the inflammatory state. The nature of this alteration in the first instance we cannot discern: its later characters are all indicative of defective nutrition; but of these, such as softening and disintegration, aptness for absorption, ulceration, and the rest, I hope to speak in a future lecture.

Here I will only add one sentence to avoid misunderstanding. I have spoken so separately of the changes in the several conditions of nutrition, that I may have seemed to imply that inflammation may consist in the disturbance sometimes of one, sometimes of another, of these states. It is true that inflammation may have its beginning in any one of these conditions,—as in an alteration of the blood in rheumatism, in an alteration of the nervous force in irritation of the retina, in an alteration of the proper elements of the tissue in inflammation of the cornea; but probably it is never fully established without involving in error all the conditions of nutrition; and, respecting both the manner in which they may be thus all involved, and their subsequent changes, they should be studied as concurrent events, rather than as a series of events of which each stands in the relation of a consequence to one or more of those that preceded it. Nowhere more than here is the mischief evident, of trying to discern in the economy of organic beings a single chain or series of events, among which each may appear as the consequence of its immediate predecessor: most fallacious is the supposition that, starting from a turgescence and stagnation of blood in the vessels of a part, we may explain the pain, the swelling, the heat, and all the other early and consecutive phenomena of inflammation. The only secure mode of apprehending the truth in this, as in every other part of the economy of living beings, is by studying what we can observe as concurrent yet often independent phenomena, or as events that follow in a constant, but not necessarily a consequent, order.

LECTURE II.

General effects of inflammation—Cessation of the process; deliquescence and metastasis; recovery of the normal circulation of the blood—Productive effects of inflammation; the several effusions or exudations attending the process— 1. *Serous exudation—its rarity—usual existence of fibrine or corpuscles in the supposed serous effusions;* 2. *Effusions of blood—secondary and primary hæmorrhages in inflamed parts—blood-stained effusions;* 3. *Exudations of lymph—General characters of inflammatory lymph—its fibrinous and corpuscular varieties—primordial forms of the various exudation-corpuscles—mixed lymph.—Comparison of the varieties of lymph with the varieties of materials effused for the repair of injuries.—Conditions determining the fibrinous or corpuscular character of the inflammatory product, and the primary tendency of the inflammation to be adhesive or suppurative—respective influences of the state of the blood, the seat of the inflammation, and its degree and general character;* 4. *Exudations of mucus—their various characters, and the transformations of the mucus-corpuscles.*

THE state described in the last lecture may, without further change, cease and pass by, and leave the part apparently just as it was before the inflammation. And there are two chief modes in which this may happen—namely, by simple cessation of the inflammation, or by deliquescence, as it has been called, and by metastasis, in which, while the inflammation disappears from one part, it appears in another. So far as the inflamed part itself is concerned, I believe the changes are, in both these cases, the same, and consist in a more or less speedy return to the normal method of circulation, and the normal apparent condition of the blood, and of the nerves, the tissue itself presenting no change of structure.

I do not know that any description of the process of recovery from the inflammatory state would tell more than is implied by calling it a gradual return to the natural state,—a gradual retracing of the steps by which the natural actions had been departed from. As it has been watched by many in the frog's web, and by myself in the bat's wing, the vessels that were filled with quick-flowing blood become narrower, the streams in them also becoming slower, and less gorged with red blood-corpuscles, till the natural state is restored. The pulsating or slower streams are equalized with those about them, and gradually making their way into the stagnant columns, drive them on or disperse them. In the frog, clusters of blood-corpuscles have been seen to become detached, by a stream breaking-off portions of the stagnant blood, and then to float into the current, where, gradually, the several corpuscles disperse. So, too, in the frog and tadpole, after injury, I have seen fragments of fibrine, washed from the blood that had clotted in the vessels of the injured part, floating in some distant vessels; but I have seen no such changes in the warm-blooded animals.

It may be difficult to explain this recovery in the case of complicated inflammations. When a slight mechanical stimulus has been applied, and the vessels, after contracting, have dilated, we may see some signs of weakened muscular power, in the fact that the same stimulus will not make them contract again; and then their gradual recovery may be the consequence of their regaining their weakened and exhausted power, just as a wearied muscle does when left at rest. This must always be one element in the recovery of the natural state by a part that has been inflamed; indeed, it is probably that part of recovery which is most slowly achieved. Still, it is, probably, only one element in the process of recovery. In an inflammation in which all the conditions of nutrition are at fault, each must recover its normal state; but, of the manner in which they severally do so, we have no other knowledge than is implied in the general truth, that all living things, after being disturbed from their normal mode of action, tend to recover it as soon as the disturbing force is withdrawn. The order in which the several disturbed conditions of nutrition will be restored is scarcely less uncertain: probably it is not constant, but may depend, in great measure, on the order in which they were involved in error. But we have no clear facts in this matter; only, we may observe, that in many cases, if we correct the error of one of the conditions of nutrition, the rest will be more apt to correct themselves. Thus, of the remedies for inflammation, few can act

upon more than one of the conditions on which it depends; yet they may be remedies for the whole disease; for, as it were, by abstracting one of its elements, they destroy the consistence and mutual tenure of the rest.

The process of deliquescence, or the mere cessation of the disease, may be regarded as the most perfect cure of which inflammation admits. It is in many cases an unalloyed advantage; but in some it is not so, though the local change may be the same; for materials accumulated in the stagnant blood of the inflamed part, or absorbed from its morbidly altered tissues, may, when the inflammation subsides, pass into the general current of the blood and infect its whole mass, or disturb the nutrition of an organ more important than that which they have left. Such are the events in the metastasis of gout, and the premature subsidence of cutaneous eruptions.

To pass now to the effects of inflammation—to the events to which the inflammatory condition may lead when it does not subside in the manner just described. They are very numerous; but they may be divided into two chief classes:—the *productive* and the *destructive* effects of inflammation. The description of the former will include the histories of the several effusions or exudations from the blood-vessels into the inflamed part, their developments, degenerations, and other changes. In the account of the destructive effects may be comprised that of the various effects of nutrition, the degeneration, absorption, ulceration, and death, to which the proper elements of the inflamed part, and, with them, the products of the inflammation, are liable.

I proceed, then, to these histories; and first, of the *products of inflammation* or *inflammatory exudations*.

The materials that may be effused from the blood-vessels of inflamed parts are chiefly these—serum; blood; lymph, or inflammatory exudation, especially so-called; and mucus; or, rather, these last two are primary forms from which, by development or degeneration, many others may be derived.

I. *The effusion of serum*, except as the result of the lowest degrees of inflammation, or as a diluent of other products, is probably a rare event. That which is usually regarded as a serous effusion in inflammation, is, in many cases, a fluid that contains fibrine, and resembles the *liquor sanguinis* rather than mere serum. It is this kind of effusion on which Vogel* has fully written, under the designation of *Hydrops fibrinosus*. A good example of it may be seen in the fluid contained in blisters, raised by the action of cantharides or heat applied to healthy persons. And another form of liquid effusion differs from serum, in that, though it does not coagulate, it contains a material capable of organisation into cells: such is the fluid that fills the early vesicles of herpes, eczema, and some other cutaneous diseases.

The fluid that contains fibrine, and is most generally described as a serous effusion, may have the ordinary aspect of serum; more rarely it is colourless or opalescent, like the liquid part of the blood which one sees collecting for the formation of a buffy coat. The fibrine that it contains may remain in solution, or without coagulation, for an indefinite time within the body, but will coagulate readily when withdrawn. For example, the so-called serous effusion, which is abundant in the integuments near the seat of an acute inflammation in deeper parts, and which flows out like a thin yellowish serum after death, will soon form a soft jelly-like clot, that is made succulent with the serum soaked in it. The fibrine appears tough, opaque-white, and stringy, when the fluid is expressed from it, and shows all the recognised characters of the fibrine of the blood. Thus, to mention but one case which was remarkable for the delay of the coagulation: a man received a compound fracture of the leg, and it was followed by phlegmonous inflammation and abscesses up the limb. As soon as the inflammation had subsided enough, the limb was amputated; and, three days afterwards, in examining it, a quantity of serous-looking fluid oozed from the cut through the integument. I collected some of this, and it formed a perfect fibrinous clot; yet the fibrine in this case had remained among the tissues without coagulating, for three days after the death of the limb, and for many more days during the life of the patient.

Such, too, are the effusions like serum in blisters raised on the skin by heat or cantharides; such the serous effusions of peritonitis, as in hernia, and of many cases of pleurisy and pericarditis. All these fluids, though they may retain their fluidity for weeks or months within the body, during life, may yet coagulate when they are removed from the body. With these, too, may be reckoned, but as the most nearly serous of the class, the fluid of common hydrocele; for I have seen a small coagulum form in such fluid spontaneously; and the presence of fibrine may always be proved by the formation of a clot, when a small piece of blood-clot, or of some

* Pathologische Anatomie, p. 23.

organized tissue is introduced into the fluid.

One can rarely tell why the coagulation of the fibrine in these cases should be delayed: there are, here, the same difficulties as are in all the exceptions from the general rules of the coagulation of the blood. But, it may be observed, the delay of the coagulation is a propitious event in all these cases of effusions; for, so long as the effusion is liquid, absorption may ensue on the subsidence of the inflammation; but absorption is more unlikely and tardy when the fibrine has coagulated. Thus, large quantities of effused fluid, which, we may be sure, contained fibrine, may disappear by absorption from the seats of acute rheumatism or gout, or from the pleura or peritoneum, or from the subcutaneous tissues, and leave only inconsiderable adhesion, or thickening of the affected part. But, on the other hand, when, in the same class of cases, the fibrine coagulates, it may be organised, and the usual consequent phenomena of inflammation will ensue. Thus it is in the cases of what has been called solid œdema (as in this specimen*), where, in the neighbourhood of acute inflammation, an effusion long abides with all the characters of ordinary serous œdema; but, at length, the tissues are found indurated and adhering, the œdema having consisted in the effusion of serum with fibrine, which has coagulated and become organised in the seats of its effusion. Thus, too, it is that the damage done by rheumatism in a part is, on the whole, in direct proportion to the length of time it has subsisted there, and the opportunity given by time for the coagulation of the fibrine.

From what I have said, it will appear that nearly all of what are called serous effusions in inflammation are effusions of fluid containing either fibrine or a material that will organise itself into cells. But it may be said that we often find effusions after death which contain nothing but the constituents of serum, though produced in an inflammatory process. If, however, we examine these cases more closely, they will appear consistent with the others: some of the fluids will coagulate if kept for several hours, or if mixed with other serous fluids, or if fragments of fibrine be placed in them; in others we find reason to believe that fibrine has been already coagulated, or that corpuscles have been formed, but that subsequently they have been disintegrated, or even partially dissolved; and in some we may believe that similar materials have been decomposed in the last periods of life, or after death.

On the whole, it seems sure that an effusion of serum alone is a rare effect of inflammation, and that generally it is characteristic of only the lowest degrees of the disease. Among the instances of it are, probably, the cases of the chronic forms of hydrops articuli, some forms of hydrocephalus, and some cases of inflammatory œdema of the mucous membrane, as in the œdema of the glottis, and chemosis of the conjunctiva.

In the nearly constant fact of the presence of organisable materials in the products of inflammation, we have one evidence of the likeness between inflammation and the normal process of nutrition, and of its difference from the merely mechanical obstructions or stagnations of the blood. In these, the material effused from the blood is usually the merely serous part: the fluids of anasarca and ascites will not coagulate; they present neither fibrine nor corpuscles, except in the cases of extremest obstruction, when, as in cases of ascites from advanced disease of the heart, one may find flakes of fibrine floating in the abdomen, or masses of it soaked and swollen-up with serum.*

II. The second of the so-called inflammatory effusions is Blood. Among the effusions of blood that occur in connection with the inflammatory process, many, as Rokitansky has explained, are examples of hæmorrhage from rupture of the vessels of lymph recently become vascular. The new vessels, or their rudiments, are peculiarly delicate; and being apt to rend, like the vessels of new granulations, with a very small force, especially when they are made turgid or dilated by an attack of inflammation of the lymph, they will commonly be sources of considerable bleeding. So, for example, it probably sometimes happens when, as the expression is, a hydrocele is converted into a hæmatocele; some lymph becoming vascular, and being submitted to even slight violence, its vessels break, and blood is poured into the sac.

* No. 2267 in the Museum of the College.

* It has been supposed that, in mechanical dropsies, the effusion of serum takes place through the walls of the small veins, and that in inflammations an equally mechanical effusion of liquor sanguinis takes place through the walls of the capillaries and small arteries; and this supposition is assumed for an explanation of the difference between a dropsical and an inflammatory effusion. But I think that in a merely mechanical obstruction of the blood, as by disease of the heart, or compression of veins, the pressure of the blood cannot but be increased alike in the veins, capillaries, and arteries, and that, in correspondence with this uniformly diffused pressure, the increased effusion will take place at once through all these vessels, in direct proportion to the permeability of their walls.

So, too, probably, it is with many or all the cases of what are called hæmorrhagic pericarditis. But of these, which may be called *secondary hæmorrhages*, I will speak hereafter.

Primary effusions of blood,—*i. e.* effusions of blood poured from the ruptured vessels of the inflamed part, and mingled with the lymph or other inflammatory product, appear to be rare in some forms or localities of inflammation, but are almost constant in others. Thus,—*e. g.* in pneumonia, extravasated blood-corpuscles give the sputa their characteristic rusty tinge. In the inflammatory red softening of the brain, blood is also commonly effused; and the condition of the vessels, which I described in the last lecture, may well account for their occasional rupture. There are also other cases of these effusions of blood in inflammation; but I believe these imply no more than accidents of the disease.

We must not confound with hæmorrhages the cases in which the inflammatory products are merely blood-stained,—*i. e.* have acquired a more or less deep tinge of blood, through the oozing of some dissolved colouring matter of the blood. The natural colour of inflammatory exudations is greyish or yellowish-white, and, even when they have become vascular, their opacity in the recent state prevents their having any uniform tint of redness visible to the naked eye. When inflammatory products present the tinge of redness, it is either because of hæmorrhage into them, or because they have imbibed the dissolved colouring matter of the blood: and when this imbibition happens during life, or soon after death, it is important, as implying a cachectic, ill-maintained condition of the blood, in which condition the colouring matter of the corpuscles becomes unnaturally soluble. Thus blood-stained effusions are among the evil signs of the products of inflammation during typhus, and other low eruptive fevers, in syphilitis, and in scurvy.

III. Serous effusions, then, appear to be rare as the results of inflammation; and effusions of blood are but accidents in its course. The characteristic primary product of the inflammatory process is *lymph*, which, in one or other of its forms or modifications, may be found as the result of inflammation in all parts.

We might wish that we had for this substance, as effused in inflammation, some other name than one which is already employed for more than one other substance —as for lymph which is contained within the lymphatic vessels, and (by some) for the liquid part of the blood. Yet, on the whole, since all these fluids—the lymph of inflammation, the lymph of the lymphatic vessels, and the liquid part of the blood—have many, and their most striking, properties in common, it may be better to retain the term lymph for them all, only specifying that which appears as the product of the inflammatory process by calling it "inflammatory" lymph. Thus used it may imply the most frequent, or even the especial, form of inflammatory exudation. The general meaning of the term may be—a liquid effused from the blood-vessels, especially from the capillaries,* which is capable of spontaneously solidifying or organising itself, even while its external circumstances remain apparently the same, and of which the parts thus solidified or organised may proceed by development to the construction of tissues.

The form assumed by lymph in its primary organisation is not always the same; there are, rather, two chief forms of organisation, which, though they are often seen mixed in the same lymph, are yet so distinct as to warrant the speaking of two varieties of inflammatory lymph by the names of *fibrinous* and *corpuscular*.†

To the fibrinous variety belong, as typical examples, all the instances in which inflammatory lymph, effused as a liquid, coagulates into the solid form, and yields, when the fluid is pressed from the solid part, either an opaque-whitish, elastic substance having the general properties of the clot of blood, or the softer, and, as it is supposed, the less perfect or less developed fibrine of the chyle or the absorbed lymph. Such examples of nearly pure fibrinous inflammatory lymph are found, in the cases already referred to, among what have been supposed to be effusions of mere serum. Such are many instances of effusions produced by blisters and other local irritations of the skin in healthy men: such, too, are most of the effusions in acute inflammations of serous membranes, especially in those of traumatic origin, and in those that occur in vigorous men. If, in any of these cases, the lymph be examined after coagulation or deposit in the solid form, it may be hard to distinguish it from the fibrine of the clot of blood. The layers of fibrinous lymph thus formed may be known to the naked eye, when on serous membranes, by their peculiar elasticity and toughness, their compact and often laminated structure, their greyish or yellowish-white and semi-transparent aspect, and their close adhesion to the membrane, even before they have become vascular.

* Or perhaps, only from them: see a remarkable case by Mr. Bowman, Lectures on the Eye, p. 44.

† Corresponding varieties are distinguished or implied by Vogel, p. 30, Dr. Andrew Clark (Medical Gazette, vol. xlii. p. 286) and others.

In the corpuscular variety of lymph, no coagulation, in the ordinary sense of the word, takes place; but corpuscles form and float free in the liquid part. Typical examples of this form are found in the early-formed contents of the vesicles of herpes, eczema, and vaccinia; in the fluid of blisters raised in cachectic patients; in some instances of pneumonia; and in some forms of inflammation of serous membranes.

The corpuscles, exudation-corpuscles, or exudation-cells, found in such lymph as this, present very numerous varieties in their several developments and degenerations; but in their first appearance resemble very nearly the primordial condition of the corpuscles of chyle and absorbed lymph, and the white corpuscles of the blood.

I may remark here, that, in this fact of a single primordial form existing in the rudiments of many organisms, which in later periods of their existence are widely different, we find a repetition of an important fact in the first development of beings. In the early embryo, even when already some traces of the future shape and construction are dimly drawn, the whole mass may be composed of cells which are all alike in their chief characters; alike, that is, in what is visible, though potentially so different, that while one group are being transformed into blood-cells, others are coalescing to form muscular fibres, others are issuing processes to form the primordial blood-vessels or lymphatics. All these, and other equally different ultimate forms, are developed from the nearly uniform mass of primordial embryo- or germ-cells. And so it is in later life; many of both the normal and the morbid structures start from one primordial form, and, thence proceeding, diverge more and more widely in attaining their several perfect shapes.

Of this primordial form we have perhaps the best instances in the white corpuscles of the blood, and in the corpuscles of lymph in the lymphatic vessels and thoracic duct. The same form is characteristic of the corpuscles in the vesicles of herpes and eczema, and in some forms of mucus. In all these, the first discernible organic form, the form of what might with propriety be called the *primordial cytoblast*, is that of a minute mass of soft, colourless, or pale greyish-white substance, round or oval, pellucid, but appearing, as if through irregularities of its surface, dimly nebulous or wrinkled. It does not look granular, nor is it formed (as many suppose) by an aggregation of granules; nor, in its earliest state, can any cell-wall be clearly demonstrated, or any nucleus. But, as the development of this cell-germ or cytoblast proceeds, a pellucid membrane appears to form as a cell-wall over its whole surface; and now, when water is added, it appears to penetrate this membrane, raising-up part of it like a clear vesicle, while upon the other part the mass retreats, or subsides, and appears more nebulous or grumous than before. In yet another state, which appears to be a later state of development, the action of water not only raises up a cell-wall, but breaks-up and disperses the outer part of the contents of the cell, *i. e.* of the enclosed mass of the primordial cytoblast, and exposes in its interior a nucleus, which is commonly round, clearly defined, pellucid, and attached to the cell-wall.

To such a nucleated cell, if we name the corpuscle from which it is developed the *primordial cytoblast*, or *cell-germ*, we may assign the name of *primordial cell*. From its various developments are derived, in the products of inflammation, all the several forms of corpuscles that are described as plastic cells, fibro-cells, caudate or fibro-plastic cells, and some forms of filaments; while, from its various degenerations, descend those known as pus-corpuscles, granule-cells, granule-masses, inflammatory globules, and much of the molecular and debris-like matter that makes inflammatory effusions turbid.

The examples of lymph which I have quoted are chiefly such as may be considered typical of the two varieties: the first, in which, spontaneously coagulating, it presents fibrine, either alone or mingled with very few corpuscles; and the second, in which corpuscles are developed alone, or with only a minimum of fibrine. But, in a large number of examples of inflammatory lymph, the fibrine and the corpuscles occur together, mixed in various proportions, the one or the other preponderating. Such instances of mixed lymph are found in the fluid of blisters in all persons not in full health; in all but the freshest inflammations of serous membranes; in most of the inflammatory deposits in cellular tissue, and in most of the viscera; and in the false membranes of croup and other similar inflammations of mucous membranes.

Now, in general, and in the first instance, the proportions of fibrine and of corpuscles that are present in the lymph of an inflammation will determine the probability of its being organised, or of its degenerating. The larger the proportion of fibrine in any specimen of inflammatory exudation (provided it be healthy fibrine), the greater is the probability of its being organised into tissue; such as that of adhesions, indurations, and the like. On the other hand, supposing the other conditions for developement or degeneration to be the

same, the larger the proportion of corpuscles in lymph, the greater is the probability of suppuration or some other degenerative process, and the more tardy is any process of developement into tissue. In other words, the preponderance of fibrine in the lymph is generally characteristic of the "adhesive inflammation;" the preponderance of corpuscles, or their sole existence, is an essential feature of the "suppurative inflammation.*

The knowledge of this fact may help us to learn the several conditions on which, in the first instance, depend these two forms of inflammation, the contrast between which has lost none of its importance since the time of Hunter. I will therefore at once enter on this question;—what are the conditions that determine the production of one or the other variety of lymph,—the fibrinous, which is as the symbol of the adhesive inflammation, or the corpuscular, which may be that of the suppurative inflammation?

The conditions, then, which are chiefly powerful in determining the character of lymph, are three; namely—

1. The state of the blood;
2. The seat of the inflammation;
3. The degree and character of the inflammation.

First, in regard to the influence of the state of the blood in determining the characters of an inflammatory product, Rokitansky has happily expressed it by saying that "the product of the inflammation exists, at least in part, in its germ preformed in the whole blood." Some, indeed, have supposed that lymph is only the liquor sanguinis exuded through the walls of the blood-vessels; but of this opinion we cannot be sure, and many facts will not agree with it. Still, it is not difficult to show that a certain character is commonly impressed by the state of the blood on the inflammatory product from it.

I will not refer here to the cases of inoculable diseases, in which some of the morbid material that was in the blood may be incorporated with the product of a local inflammation, though in these the correspondence of the blood and the inflammatory product from it is manifest enough; but I will refer to cases that may show a more general correspondence between the two, a correspondence such that, according to the state of the blood, so is the lymph more fibrinous or more corpuscular; more characteristic of the adhesive, or of the suppurative inflammation.

Some of the best evidence for this is supplied by Rokitansky, in the first volume of his Pathological Anatomy; a work that I cannot mention without a tribute of respect and admiration for its author, since in it, more than in any other of his writings, he has proved himself the first among all pathologists,—in knowledge at once profound, minute, and accurate, in power of comprehending the vastest catalogue of single facts, and in clear discernment of their relations to one another, and to the great principles on which he founds his systems. In this work, he has shown clearly, that the characters of inflammatory deposits in different diatheses correspond very generally and closely with those of the coagula found in the heart and pulmonary vessels after death; and that, in general, the characters of lymph formed during life will be imitated by those of clots found in the body after death, when the fibrine of the blood may coagulate very slowly, and in contact with organic substances.

Other evidence may be obtained by examining the products of similar inflammations excited in several persons, in whom the state of the blood may be considered dissimilar; and here the evidence may be more pointed than in the former case; for, if it should appear that the same tissue, inflamed by the same stimulus, will, in different persons, yield different forms of lymph, we shall have come near to certainty that the character of the blood is that which chiefly determines the character of an inflammation. To test this matter, I examined carefully the materials effused in blisters raised by cantharides-plaisters

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* In this view, the fibrinous and the corpuscular varieties of lymph nearly correspond with those which Dr. Williams, in his Principles of Medicine, and others, have named plastic and aplastic; but they do not completely do so. In different instances of both varieties, very diverse degrees of plastic property may be found; and the occurrence of development or degeneration depends on many things besides the primary characters of lymph. They more nearly correspond with what Rokitansky (Pathologische Anatomie, i., 196) has distinguished as fibrinous and croupous; the varieties which he names croupous α, β, and γ, representing the several grades of lymph in which the corpuscles gradually predominate more and more over the fibrine, and assume more of the characters of the pus-cell. I would have used his terms, but that, in this country, we have been in the habit of considering croupous exudations to be peculiarly fibrinous.

In the last course of Lectures (Medical Gazette, Vol. xliii.), I described the healing of subcutaneous wounds as usually accomplished by a fibrinous material, and that of open wounds by cells developing into fibres. These materials exactly correspond in appearance and modes of development with the fibrinous and corpuscular varieties of inflammatory lymph; and I was quite wrong in implying that fibrine alone is not a product of inflammation, and that all the filamentous tissue formed after inflammation is developed through cells. What was then said, however, of the liability of the cells formed in the repair of open wounds to be arrested in their development, or to degenerate into pus-cells and lower forms, and of the consequent insecurity of this mode of repair as compared with the subcutaneous, is confirmed by the corresponding history of the two varieties of lymph.

applied to the skin of thirty patients in St. Bartholomew's Hospital. Doubtless, among the results thus obtained, there might be some diversities depending on the time and severity of the stimulus applied; still, it seemed a fair test of the question in view, and the general result proved it to be so. For, although the differences in the general aspects of these materials were slight, yet there were great differences in the microscopic characters; and these differences so far corresponded with the nature of the disease, or of the patient's general health to whom the blister was applied, that at last I could generally guess accurately, from an examination of the fluid in the blister, what was the general character of the disease with which the patient suffered. Thus, in cases of purely local disease, in patients otherwise sound, the lymph thus obtained formed an almost unmixed coagulum, in which, when the fluid was pressed out, the fibrin was firm, elastic, and apparently filamentous. In cases at the opposite end of the scale, such as those of advanced phthisis, a minimum of fibrin was concealed by the crowds of corpuscles imbedded in it. Between these were numerous intermediate conditions which it is not necessary now to particularize. It may suffice to say that, after some practice, one might form a fair opinion of the degree in which a patient was cachectic, and of the degree in which an inflammation in him would tend to the adhesive or the suppurative character, by the microscopic character of these exudations. The highest health is marked by an exudation of the most perfect and unmixed fibrine; the lowest, by the most abundant corpuscles, and their nearest approach, even in their early state, to the characters of pus-cells. The degrees of deviation from general health are marked, either by increasing abundance of the corpuscles, their gradual predominance over the fibrine, and their gradual approach to the character of pus-cells, or else by the gradual deterioration of fibrine, in which, from being tough, elastic, clear, uniform, and of filamentous appearance or filamentous structure, it becomes less and less filamentous, softer, more paste-like, turbid, nebulous, dotted, and mingled with minute oil-molecules.

I would not make too much of these observations. They are not enough to prove more than the rough truth, that the products of similar inflammations, excited in the same tissue, and by the same stimulus, may be in different persons very different, varying especially in accordance with the several conditions of the blood. Yet, simple as the observations are, they may illustrate what often seems so mysterious—namely, the different issues of severe injuries inflicted on different persons. To what, more than to the previous or some acquired condition of the blood, can we ascribe in general the various consequences that follow the same operations in different patients? The local stimulus, and the conditions by which the inflammatory effusion finds itself surrounded, may be in all alike; but, as in the simpler case of the blister, the final events of the inflammation are according to the blood.

I cannot doubt that a yet closer correspondence between the blood, and the products of inflammation derived from it, would be found in a series of more complete observations,—in such, for instance, that the characters of the blood drawn during life, or, much better, of the clots taken from the heart after death, might, in a large number of patients, be compared with those of inflammatory exudations produced, as in the cases I have referred to, by the same stimulus applied to the same tissue. In the few cases in which I have been able to make such examinations, this view has been established; and it is confirmed by the parallelism between the varieties of lymph that may be found in blisters and the varieties of the fibrinous coagula in the heart described by Rokitansky.* The varieties of solidified fibrine which he enumerates and classifies as fibrines 1, 2, 3, 4, are very nearly parallel with what I have enumerated as the stages from the best fibrinous to the corpuscular lymph; and, as I have already implied, he regards these clots found in the heart and vessels as representing the different "fibrinous crases" or diatheses of the blood.

I mentioned, as the second condition determining the issue of an inflammation, the seat or tissue which it occupies. I need hardly remind you that, since the time of Bichat, it has been a general impression that each tissue has its proper mode and product of inflammation. The doctrines of Bichat on this point were, indeed, only the same as Mr. Hunter held more conditionally, and, therefore, more truly; but they gained undisputed sway among the principles of that pathology which rested on general anatomy as its foundation.

The facts on which it is held that, in general, each part or tissue is prone to the production of one certain form of inflammatory exudation, are such as these,—that, *e. g.*, in the apparently spontaneous inflammations of the skin, lymph with corpuscles alone is produced, as in herpes, eczema, erysipelas; that in serous mem-

* Pathologische Anatomie, B. i. p. 142.

branes, the lymph is commonly fibrinous, and has a great tendency to be organised, and form adhesions; that in mucous membranes there is as great a tendency to suppuration; that in the lungs, both fibrine and corpusles are abundant in the lymph, and the corpuscles have a remarkable tendency to degenerate into either pus-cells or granule-cells; that in the brain and spinal cord the tendency is to the production of a preponderance of corpuscles, that quickly degenerate into granule-cells; while in the cellular tissue, both fibrine and corpuscles appear, on the whole, equally apt to degenerate into pus, or to be developed into filamentous tissue.

Now these are, doubtless, facts; but the rules that it is sought to establish from them are not without numerous exceptions. The instances I have lately quoted show that, in one tissue at least, the skin, the products of inflammation will vary according to the condition of the blood, although the inflammation be always similarly excited by the same stimulus. So, too (as Mr. Hunter remarks*), if it were the tissue alone that determines the character of an inflammation, we ought to have many forms of inflammation in the same stump after amputation; whereas all is consistent, or the differences among the tissues are only differences of degree: they all adhere, or all granulate and suppurate, or all alike inflame or slough.

It is therefore not unconditionally true that each tissue has its proper mode and product of inflammation. It has been too much overlooked that a morbid condition of the blood, or perhaps even of the nervous force, may determine at once the seat of a local inflammation, and the form or kind of inflammatory product. Thus, *e. g.*, the variolous condition of the blood may be said to determine at once an inflammation of the skin, and the suppurative form of inflammation; for, in variola, whatever and wherever inflammations arise, they have a suppurative tendency. So, in rheumatism, whether it be seated in muscles, ligaments, or synovial membranes, in serous membranes, or in fibrous tissues, there appears the same tendency to serous and fibrinous effusions, which are slow to coagulate or organise, and even less prone to suppuration. And so in the purulent diathesis; in whatever tissue the malady of the blood may localise itself, the tendency of the inflammatory product is to the formation of pus. The same might be said of the local inflammations that are characteristic of typhus and of gout, and, I believe, of all those diseases in which a morbid condition of the blood manifests itself in some especial local error of nutrition. And all these cases are illustrative of the general truth, that each morbid condition of the blood is prone both to produce an inflammation in a certain part or tissue, and to give to that inflammation a certain form or character.

Cases, however, remain that prove some influence of the tissue in determining the product of its inflammation—in determining, I mean, the primary form, as well as the later development, of the product: and the true influence of the tissue in this respect is best shown in some of the cases in which inflammation, excited, apparently, by the same means, has happened coincidently in two or more very different parts in the same person. Thus we may find, *e. g.*, that in pleuro-pneumonia the lymph on the pleura is commonly more fibrinous than that within the substance of the lung; and adhesions may be forming in the one, while the other is suppurating. In cases of coincident pneumonia and pericarditis, the lymph in the lung may appear nearly all corpuscular, and all the corpuscles may show a tendency to degenerate into granule-cells, while the lymph on the pericardium may have a preponderance of fibrine, and what corpuscles it has may tend to degenerate into pus-cells. So, too, one may find, in the substance of an inflamed synovial or mucous membrane, abundant lymph-cells, while all the exudation on its surface may appear purulent.

Other instances of this kind might be cited,—enough to establish that the nature of the tissue or part affected has some share in determining the character of the products of its inflammation. On the whole, therefore, the best conclusion may be that, in any given case, the issue of an inflammation, and especially as to whether it will correspond with the adhesive or suppurative form, may be represented as the resultant of these two chief influences—the previous condition of the blood, and the nature of the part affected.

I will only add, that the supposed influence of a tissue in determining the character of the lymph formed in its inflammations, may be very probably explained by believing that the primary product of inflammation is, often, a mixture of lymph, and of the secretion, or other product of the inflamed part, more or less altered by the circumstances of the inflammation. When it is seen that in inflammations of bone the lymph usually ossifies,—in those of ligament is converted into a tough ligamentous tissue,—and that, in general, lymph is organised into a tissue more or less corresponding with that from whose vessels it was derived,—it is usually concluded that this happens under what is called the assi-

* Works, vol. iii. p. 813.

milative influence of the tissues adjacent to the organised lymph. But it seems more probable that no such assimilative force is exercised after the effusion; rather, we may explain the facts by believing that the material formed in the inflammation of each part partakes, from the first, in the properties of the natural products of that part; in properties which we know determine the mode of formation independently of any assimilative force.

We have some evidence of this in the products of inflammation of secreting organs, the only structures of which we can well examine the natural products in their primary condition. In a moderate amount of inflammation of a secreting gland, the discharge is usually a mixture of the proper secretion in a more or less morbid state, and of the inflammatory product. Thus we find morbid urine mixed with fibrine, or albumen, or pus. In cases of inflamed mucous membranes, the product is often a substance with characters intermediate between those of the proper mucous secretion and those of lymph. Or, again, in some membranes we may perceive a relation between their natural secretion and the usual products of their inflammation.

With these considerations I may connect what is to be said of the influence of the third among the conditions enumerated as determining the charater of any inflammatory product—namely, the degree or severity of the disease. For, as a general rule, the less the degree of inflammation is, the more is the product like that naturally formed in or by the part, till we descend to the border at which inflammation merges into an exaggerated normal process of secretion; as in hydrops articuli, hydrocele, coryza, &c.

The analogies between secretion and nutrition are so numerous, the parallel between them is so close, that what can be shown of one may be very confidently assumed of the other. We may therefore believe that, in the inflammation of any part, the product will, from the first, have a measure of the peculiar properties of the material employed in the normal nutrition of the part: that, as in the inflammation of a secreting organ, some of the secretion may be mingled with the product of the inflammation, so in that of any other part, some of the natural plasma—*i. e.* some of the material that would be effused for the healthy nutrition of the part—may be mingled with the lymph. The measure of assimilation to the natural structure will bear an inverse proportion to the severity of the inflammatory process, because, the more the conditions of nutrition deviate from what is normal, the more will the material effused from the vessels deviate from the normal type. In severest cases of inflammation we may believe that unmixed lymph is produced, the conditions of the due nutrition of the part being wholly suspended; but when the inflammation is not altogether dominant, its product will be not wholly contrary to the natural one, and will, from the first, tend to manifest in its development some characters correspondent with those of the natural formations in the part. Thence, onwards, this correspondence will increase as the new tissue is itself nourished: as scars improve, so do false membranes and the like become more and more similar to natural tissues.

To sum up, then, what may be concluded respecting the conditions that, in the first instance, may determine the adhesive or suppurative characters of an inflammatory exudation; they are—1st. The state of the blood—its diathesis or crasis—the power of which is evident in that the same material may be exuded in many inflamed parts in the same person; in that this material may exhibit peculiar characters correspondent with those of the blood itself; and in that, in different persons, an inflammation excited in the same tissue, and by the same stimulus, will produce different forms of lymph, corresponding with differences of the blood. 2d. The seat of the inflammation, and the tissue or organ affected, of which the influence is shown by cases in which, with the same condition of blood, different exudations are produced in different parts or organs. 3d. The severity, and acute or chronic character, of the inflammatory process, according to which the product deviates more or less from the character of the natural secretion or blastematous effusion of the inflamed part.

The primitive character or tendency of any case of inflammation might be represented as the resultant of three forces issuing from these conditions.

The last product of inflammation of which I have to speak is *Mucus*.

Peculiar difficulties, owing to imperfect investigations of what normal mucus really is, beset this portion of our subject. I will only venture to say that—

1. Normal mucus is a peculiar viscid, ropy, pellucid substance, which, of its own composition, has no organised particles. Such mucus is to be found in the nasal cavities of sheep and most large mammalia, in the human uterus, and in the gall-bladder when its duct has been lately obstructed. In all these mucus may be found without corpuscles; and probably there are other examples of such pure and unmixed mucus.

But 2d. With all these, accidental mixtures commonly occur of epithelial parti-

cles from the mucous membrane. And these particles will vary according to the seat of the membrane; the fluid with which the mucus may be mixed, as gastric acid, intestinal alkali, &c.; the time the mucus may lie before discharge; and other such conditions.

3. The first effect of a stimulation, within the normal limits, will be to increase the secretion of the proper mucus, making it also more liquid—to increase the quantity of the epithelium cast-off with the liquid—and, often, to induce the premature desquamation of the epithelium, so that particles of it imperfectly formed may be found in the mucus. Many of these immature epithelial particles have been named mucus-corpuscles, or mucus-cells.

4. In an established inflammation of a mucous membrane, there appear, in greater or less proportion mixed with mucus, materials which have naturally no part in it, and which may be closely paralleled, or considered identical, with the products of inflammation in other parts. I am, indeed, disposed to think that we should not draw a strong contrast between the inflammatory products of mucous membranes and those of serous membranes and other parts, except in relation to the material with which, in the several cases, the inflammatory lymph is mixed. For in certain inflammations of mucous membranes we find fibrinous exudations, as in Hunter's experiments of injecting strong irritants into the vaginæ of asses*; in probably less pure forms in croup and bronchial polypus†; and, as I have seen it, in the renal pelvis, ureters, and bladder in a case of calculus In other cases, we find corpuscles, which appear to differ from those in the exudations already described only because of the peculiarly viscid fluid in which they lie. All are, alike, lymph-corpuscles: but in the one case they lie in a serous, in the other in a mucous fluid.

5. From these inflammatory products may be derived, by various degenerations of the fibrine, the flaky and molecular materials which commonly make morbid mucus look turbid and opaque; and by corresponding degenerations of the corpuscles—*i. e.* of the lymph-corpuscles, not of any normal cells or nuclei—the more frequent pus-cells, which make the transition to the pure pus secreted by mucous membranes in active inflammation.

Such degenerations are more frequent in the products of inflamed mucous surfaces than are any forms of development. Development of fibrine, I suppose, never happens here; but in the corpuscles some indications of it may be found, especially when the inflammation is very slight, as in the end of a bronchitis. If the grey, smoke-coloured mucus expectorated at this time be examined, it will be found that the peculiar colour, though commonly ascribed to the mixture of inhaled carbon, is due to the abundance of cells containing more or less numerous black pigment-granules. Particles of carbon or soot may by chance be present, but they only trivially contribute to the colour: it depends on the number of these pigment-cells, to which it is easy to trace the transitions from lymph or mucus-corpuscles. The chief stages of transition are seen in that the cells enlarge, become clearer, and acquire one or two clear oval nuclei; but, at the same time, minute black granules, almost like those of melanotic cells, accumulate in them; and these, increasing in number and clustering, may at length fill the whole cell, while the nucleus disappears. Subsequently the cell-wall may burst or dissolve, and the black granules be set free.

It may not be supposed that the black granules are in any way derived from inhaled carbon, although it seems that this kind of mucus is most abundant in those who are exposed to atmospheres laden with coal-smoke; for the colour is completely destroyed by immersing the mucus in nitric acid or solution of chlorine. The occurrence of such pigment-cells being, I believe, peculiar to the mucus of the air-passages, may be connected with the general tendency of inflammatory products to imitate the properties of the natural products of the inflamed part; for they closely resemble the black pigment-cells from which the lungs and bronchial glands derive their black spots and streaks and other marks. And, it may be added, that their peculiar abundance in the slighest forms of bronchitis, compared with their absence in acute cases, affords another example that the likeness of the morbid to the natural product is inversely proportionate to the severity of the inflammation.

* Works, vol. iii. p. 269, 341. Museum of the College, Nos. 83, 84.

† See Henle, in his Zeitschrift, T. ii. p. 178.

LECTURE III.

Developments of the lymph-products of Inflammation—General meaning of 'development;' limitations with which the term is to be used in regard to some tissues—Conditions of its occurrence in lymph—Necessity of the cessation of the inflammation — Conditions determining the direction of the development—Respective influences of the nature of the lymph itself, and of the tissue in or near which it is placed—Participation of the lymph in the properties of the normal blastema of the inflamed tissue—Developments into fibro-cellular, fibrous, epithelial, osseous, and other tissues—Developments of blood-vessels and lymphatics in lymph—Time requisite for development of blood-vessels.

In the last lecture I spoke of the lymph effused in inflammation as presenting certain primary forms, the fibrinous and corpuscular, from which, by their various developments and degenerations, are derived those other structures, which are characterised as products or results of inflammation; such as, through development, adhesions, indurations, and the like; through degeneration, pus, ichor, granule-cells, &c. It is in the biography of the lymph-product that much of the most important part of the pathology of inflammation is comprised: and if it were required to point out what, since Hunter's time, has contributed most to the progress of general pathology, one could scarcely hesitate to name the full appreciation of the fact, that lymph, like the other primary products of disease, has an independent life, and is of its own nature capable of appropriate development, degeneration, and disease. We may regard this as one of the best achievements of the observations which Schleiden and Schwann began to generalise; for, till it was clearly apprehended, the idea of a part being organizable meant scarcely more than that it admitted of being organized by the forces of the parts around it; that it could be built-up by the arteries, and modelled by the absorbents, as a material pliant, yet passive, in the hands of workmen. Hence was derived the erroneous direction of inquirers, which sought for blood-vessels as the essential characters of organic life in a part; and for their varieties of size, and number, and arrangement, as the measures of the ability and method of development.

But now, more truly, we may study lymph as having a life only so dependent on the blood and vessels as are all the tissues of the body—dependent on them as conditions of existence, but not as sole arbiters of the method or direction of the vital transformations. And I venture to think, that the chief aim of our observations in the pathology of inflammation should be to learn, now, the exact relation in which the several products of inflammation stand to certain primary forms, as developments or degenerations from them. The catalogue of various corpuscles is already swollen to an extent that is confusing to those who are familiar with them, and repulsive to those who would begin to study them. It would be an easy task to increase it, and it might have a seeming of accuracy to do so; but what we want, is such a history of lymph that we may arrange the components of this catalogue as indicating so many progressive stages of development, degeneration, or disease, in the primary products of inflammation. An attempt to construct such a history is the more advisable, for the sake of the illustration which it may afford to the history of normal structures. There are no normal instances in which we can see the materials that are effused for the nutrition of parts; but we may assume something concerning them and their progressive changes from the analogy of the materials that are more abundantly produced in inflammations.

I propose to devote the present lecture to some general,—and only a very general,—account of the developments of lymph. But let me first state the sense in which the term development is here to be employed. By development of a part, speaking generally, is understood "the process by which an organ or tissue is first formed; or by which one, being already imperfectly formed, is so changed in shape or composition, as to be fitted for a higher function; or, finally, is advanced to the state in which it exists in the most perfect condition of the species."* In the use of this definition we shall find that we have adopted

* See Lectures on Nutrition, &c., in the Medical Gazette, 1847.

an arbitrary standard of comparison, on the assumption that the nearest approach to organic perfection is in the human body, at the age of manhood. The assumption may be right on the whole; and a less arbitrary definition of development would, probably, be less useful; yet it may be observed, that in what we take for the period and standard of perfection, many parts that were once highly organized and active have passed away, as the thymus gland; and some are, in certain respects, rather degenerated than developed, as the renal capsules and the bones.* We cannot unconditionally call that change a development, in which a part acquires a new chemical composition less remote from that of inorganic matter, as bone does when its chondrine is replaced by gelatine, and when this becomes impregnated with abundant saline and earthy matter, and leaves spaces filled with fat. Neither is that change altogether development, in which, as in the formation of ligamentous tissue, the organic life of the material becomes less active; or which, like ossification, can be accomplished as well in the feebleness of advanced life, as in an earlier period; as well in disease as in health. Development, in its highest sense, should imply not merely that a part becomes more fit for membership under the most perfect economy, but, also, that such fitness is acquired with greater complexity of chemical composition, or with greater evidence of formative or other organic power, or with greater difference from the structure or composition of lower beings. With none of these characters of development does such a process as that of ossification agree; and, therefore, when we call it the 'development' of bone from cartilage, it should be with the understanding that the term is applicable only because bone is the proper material of the skeleton of the adult human body.

This distinction is important in the pathology of inflammation. In all true or complete development we may believe there is a larger expenditure of vital force than in any other organic act; for all such development, too, the external conditions need to be the most complete, and the least interfered with; such development is the highest achievement of the vital force—the highest instance of what might be understood as 'increased action' in a part. It is in accordance with this that, in general, development is arrested in every severe disease, and that the residual capacity of repair, and other organic processes, is, in each species, inversely proportionate to the amount of original development, or, to the distance between the embryonic and the perfect forms of the species.

To speak, therefore, of the development of inflammatory products, when already the normal development of the body is completed, may seem to imply the exercise of unusual vital force—the renewal, as it were, of the pristine embryonic vigour—and the existence of conditions more favourable for nutrition than even those of health are. But we may be led to judge differently, if it should appear that most or all of the so-called developments of inflammatory products are instances in which the tissues, though they are formed into the likeness of such as exist in the perfect human frame, yet acquire characters of lower organization than those they had in their earliest state. It will appear that they are such; and that however much the inflammatory products may become, by their changes, better suited for the general purposes of the economy, they are, in relation to their own condition, rather degenerated than developed. The changes that they undergo are, therefore, not always declaratory of a large expenditure of vital force; they are not such as the term 'sthenic,' applied to the inflammatory process, would suggest; not such as to imply that it is an exaggeration of any normal method of nutrition.

With this understanding, however, the changes I shall presently describe may be called developments of inflammatory lymph or exudation; they are developments in the sense of being approximations to the likeness of the natural tissues of the adult human body.

In the last lecture I spoke, generally, of the conditions upon which depends the production of such inflammatory lymph as may be most apt for development. They are all such as favour the production of a lymph rich in fibrine, and that fibrine clear, homogeneous, elastic, tough, and filamentous.* But even such lymph as this may altogether fail to be developed, or may be arrested in any stage of its development, and turned into the downward course of degeneration, unless favourable external conditions are present with it. For the development of lymph, of whatever form, nearly all those conditions are requisite, which are necessary for the normal development of the proper constituents of the body. It needs, in general, the due supply of healthy and appropriate

* See Lectures on Repair, in MEDICAL GAZETTE for 1849.

* When fibrine is thus spoken of, one does not refer to the chemically pure fibrine which may be obtained by art, and which, most probably, could not be organized; but to the compound in which fibrine is naturally mixed with fatty, extractive, and saline matters, all of which, we may believe, are as necessary to right development as itself is.

blood, the normal influence of the nervous force, and, for the highest and latest forms of development, the normal condition of the proper elements of the affected part.

Now the existence of these conditions for the development of lymph implies a cessation of the inflammatory process, and a recovery from whatever originated or maintained the inflammation. So long as inflammation lasts, no high development of the exudation already formed will take place; rather, fresh exudation will be continually formed, hindering the due process of development, and hindering it the more, because, as the general health suffers through the continuance of the disease, so the lymph freshly formed will be less and less prone to organization. We may see this illustrated in bad cases of pleurisy: the layers of lymph next to the pleura are always more prone to organization than the later formed layers that lie next the cavity; while within the cavity all the lymph may retain its fluid form, or may have degenerated into pus. So, more openly, we may see an illustration of the ill effects of abiding inflammation, in the healing of wounds by granulations. An inflammation ensuing or continuing in the wound hinders all development of granulation-cells, even though it may be too slight to hinder their formation, and may be favourable to the production of the ichor- and pus-cells. We may truly say, that the conditions most favourable to the production of lymph are among the most unfavourable to its development, *i. e.* to its complete and higher organization.

Even when the inflammation has ceased, and fresh exudation is not formed, still the development of the lymph is often prevented or retarded by want of some necessary condition. The blood-vessels, long dilated, may remain in a state of congestion, distended as if paralysed, and filled with slowly moving blood. In such a state—the state of 'passive congestion'—so apt to follow more acute attacks, development will not happen in even well-disposed lymph. We have parallel facts in the tardy development of granulations on the legs, as in the healing of ulcers; and how much this depends on the defective movement of the blood is well illustrated by a specimen* appropriate to an observation of Mr. Hunter's. It shews three ulcers of the integuments of a leg; they are all granulating, and all healing; but their progress in healing has been inversely proportionate to the hindrances of the blood. The lowest of the three, that most distant from the heart, and of which the vessels were subject to the pressure of the highest column of blood, is least advanced in healing; while the uppermost of the three is most advanced, and is nearly cicatrized.

But let us suppose all the conditions for development provided: what will now determine the direction or result of the process? Into what tissues will the lymph be formed? Two chief things will determine this: first, the natural tendency of organizable lymph, produced in inflammation, is to form filamentous, *i. e.* fibro-cellular or fibrous tissue; and, secondly, all lymph has some tendency to assume, sooner or later, the characters of the tissue in or near which it is seated, or in place of which it is formed.

The natural tendency of lymph to the construction of fibro-cellular or connective tissue, such as composes false membranes and adhesions, and most thickenings and indurations of parts, is shewn by the production of this tissue under all varieties of circumstances, and in nearly all parts; even in parts which, naturally, contain little or none. Thus, it is found in the brain, and in glands, as in the testicle; within joints, even where adhesions only pass from one articular cartilage to another; in the adhesions and thickenings of the most diverse serous membranes; in the thickenings of the most diverse mucous ones. And with all these, we have corresponding facts in the healing of wounds: all granulations, springing from what surface they may, tend, at least in the first instance, to the formation of filamentous tissue, such as we see uniting all parts in a stump; and a large proportion of subcutaneous injuries are repaired by similar tissue, whatever parts may have been divided. And sometimes we may find instances of this development where the lymph is not even in continuity with any tissue, but floats free; as in ascites, or in effusions into joints.

But besides this general tendency, we may recognize in lymph a disposition to assume characters belonging to the part in which it was produced; so that, for instance, that about fibrous and ligamentous parts will be developed into peculiarly tough fibrous tissue; that about bone will become osseous; that in the neighbourhood of epithelium will form for itself an epithelial covering; and so on. I referred to this fact in the last lecture. The conformity of the developed lymph to the characters of the parts around it is usually ascribed to an assimilative force, exercised by those parts after the effusion of the lymph, and during its development. But it seems more probable that the tendency to conformity is from an original and inherent quality of the lymph; the material formed

* Mus. Coll. Surg. No. 26, Catal. vol. i. p. 15.

in the inflammation of each part partaking, from the first, in the properties of the natural products of that part, properties which, we know, determine the mode of formation in the natural process of nutrition, independently of any assimilative force.

As I stated in the last lecture, the degree in which lymph will partake of the properties of the natural product of the part will bear an inverse proportion to the severity of the inflammation; because the more the normal conditions of nutrition are deviated from, the more will the material produced be unlike the normal product. And when the conditions are restored to the normal type, so will the organized product of inflammation constantly approximate more and more to the characters of the parts among which it is placed, or with which it has acquired membership. As scars improve, *i. e.* gain, gradually, more of the characters of skin, so do false membranes and the like acquire, by their own nutrition and development, more nearly the characters of the parts with which they are connected. Thus false membranes in the serous cavities acquire a covering of epithelium, exactly like that which covers the original serous membrane, and their tissue becomes perfectly fibro-cellular: thus, too, adhesions of the iris may become black, apparently from the production of pigment-cells like those of the uvea; thus, too, in adhesions of the pleura, even when they are long and membranous, pigment may be formed as in the pulmonary pleura itself;* and thus many other products are gradually perfected, till we may come to doubt whether they be of normal or of morbid origin, so complete is the return from the aberrant action.

I will endeavour, now, to describe more particularly the modes of development into the several tissues that may be formed from lymph. And here I may refer to a part of the lectures I had the honour of delivering last year, in which I spoke of the two different materials produced for the repair of subcutaneous and of open wounds. The material for subcutaneous wounds appears to be a nearly unmixed fibrine, and is comparable in appearance, though not in origin, with the best forms of fibrinous lymph: the material for open wounds, such as granulations are formed of, consists almost exclusively of cells, and is equally comparable with the corpuscular lymph of inflammation. I then also described the respective methods of development, through which these two materials are transformed into the several permanent tissues of repair; and especially described how similar fibro-cellular and fibrous tissue may be formed from them both, by different routes of development. Such tissue is formed from the fibrinous material by the production of nuclei, constituting the 'nucleated blastema,' and from the granulation-cells by their elongation and attenuation into filaments.

I believe that corresponding methods of development are observable in the two similar forms of inflammatory lymph; that the fibrinous becomes a nucleated blastema, in a stage previous to the formation of the fibro-cellular tissue; and that the corpuscular lymph is developed through the elongation of its cells.

Now these are the recognized methods by which the normal fibro-cellular and fibrous tissues are developed; that through nucleated blastema is the method described more particularly by Henle; that through the elongation and attenuation of nucleated cells is the method first described by Schwann; and, probably, where this last prevails, we may assume that, very often, while cells are elongating into filaments, the intercellular substance, which is sometimes abundant, is assuming the same filamentous character. Such a combination of the two methods of development may be assumed for all those examples of lymph in which the fibrine and the corpuscles are mingled.

I need not, at greater length, describe these well-known methods of development. It may suffice to say, that after presenting transition-stages in exact conformity with those observed in the normal development of the like tissues, the lymph of inflammation may become perfect fibro-cellular or fibrous tissue. The imitation, in adhesions of serous membranes, in the indurated ligamentous substance formed in and about muscles, tendons, and the like, is perfect. And if it should seem strange that disease should thus so closely imitate health, the explanation is, that this process in lymph is not disease. The lymph is, indeed, produced in inflammation, but it is developed in health, when all the natural conditions of nutrition are restored.

It would be tedious, and quite unnecessary, to enumerate all the particular forms in which lymph developed into fibro-cellular and fibrous tissues may present itself. The general forms are—1. adhesions, where the tissue is between free surfaces, and unites them; 2. thickening, where the formation is in the substance of membranes; 3. indurations, with or without contractions, where the tissue is formed in the substance of organs; 4. opacities of parts that were transparent. All these are minutely described in the standard works on pathology and morbid anatomy.

* As in No. 96, in the Museum of the College.

The numerous varieties of tissue that may be included in the class of fibro-cellular and fibrous tissues comprise nearly all the normal structures that can be formed from the lymph of inflammation in man. Besides these, indeed, none are certainly formed except the structures of epithelium and bone.

Epithelial cells, we know, are formed of the cells of granulations in the "skinning" of sores, and they present a striking instance of the purpose observed in the healing process; for cells are here, as with design, formed into epithelium, while cells like them, more deeply seated, are being developed into filamentous tissue. The cells of lymph are so like those of granulations, that we should be justified in believing that they also may be developed into epithelial cells, even if the opportunities of tracing the process were rarer than they are. We may sometimes find, in inflammation of serous membranes, recent lymph-cells presenting many characters indicative of development towards epithelium, flattening and enlarging, and acquiring circular or oval, clear nuclei. On old false membranes, epithelial cells are perfectly formed; but these may have been produced after the complete organisation of the membrane.

Bone is often formed from lymph. It may appear as a late transformation of lymph that has been organised into perfect fibrous tissue, as in the osseous plates that are sometimes found in the false membranes of the pleura, or in the pericardium. But in most of these there is not true bone, but an amorphous deposit of earthy matter, which is imbedded in the fibrous tissue, or which (as Rokitansky holds) is the residue of the degenerated and partially absorbed tissue.

The proper condition for the transformation of lymph into bone is that in which the exudation takes place in an inflammation seated in or near the periosteum. Such inflammations have been called "ossific;" and the museum of the College, like every other, abounds with specimens of their various results. The second and fifth volumes of the catalogue of the museum contain such detailed descriptions of these specimens, that I need only mention some general principles which they illustrate.

We have evidence enough that superficial deposits of new bone are formed as well after inflammation of the periosteum, as after inflammation of the bone itself; or even more constantly. And we might expect such a result, knowing, from Dr. Sharpey's and Kolliker's researches, that the growth of bones in circumference is accomplished by the ossification of successive layers of fibrous tissue, like the periosteum, and that such fibrous tissue is one which the developed products of inflammation may imitate. It is to the ossification of such fibrous tissue, formed (though probably only in rudiment) in periostitis, that we may ascribe the greater number of superficial formations of new bone, and more especially the thin plates of bone that are sometimes imbedded in the periosteum, or even on its exterior, and completely detached from the surface of the bone.

It is also worth observing how characteristic of different diseases are certain formations of new bone produced in inflammations. The pustules of variola, or the vesicles of herpes, are scarcely more characteristic of those diseases, than are the hard nodules of cancellous bone clustered about the articular borders of bones that have been the seat of chronic rheumatism,* or the porous, friable, dirty, and readily ulcerating thin layers formed on the shafts in syphilis,† or the circumscribed, flat elevations of bone formed under old ulcers of the integuments.‡

I am not aware that the mode of ossification which prevails in any of these cases has been minutely studied; but we may believe the ossification of inflammatory products to be essentially similar to those observable in the repair of fractures. It is probable that cartilage is very rarely, if ever, formed in inflammatory lymph; for it seems to be formed in the repair of fractures only when the conditions are more favourable than they are likely to be in any inflammations. Probably, therefore, the lymph is more or less developed towards the fibrous tissue when it ossifies; and, as in the repair of fractures, so here, we may believe that ossification may be postponed till the fibrous tissue is quite formed, or that it may ensue in the rudimental state of the tissue, whether in a nucleated blastema, or in cells like those of granulations.§

Such are the developments of which lymph appears capable in man; and I doubt whether any other tissues are ever formed from its materials. All the instances in which it has been said that transversely striated muscular fibres were formed seem questionable. The formation of the tissues of the internal membrane of bloodvessels is probably accomplished by development of fibrine-deposits from the blood flowing in the vessels whose walls are thus thickened; at least, their formation

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* As in 572, and others, in the museum.

† As in Nos. 628 to 631.

‡ As in Nos. 581 to 585, and others.

§ These subjects are more fully treated in the Lectures on Repair published last year.

from lymph deposited from the *vasa vasorum*, as in phlebitis, has not been nearly proved.

The formation of the proper bloodvessels of organising lymph is, I believe, not from the materials of the lymph itself, but by outgrowth from the vessels of the adjacent part; but on this point I must say something more, if only because different opinions are held by some of the highest authorities.

The question is, whether the bloodvessels of organising lymph are formed entirely of the material of the lymph, and, as it were, by its own power of development, or whether they are outgrowths from adjacent natural or original vessels, which, as the expression is, shoot-out into the lymph. Both these modes of formation of bloodvessels are observable in the natural tissues while they are in process of development; the new vessels which are formed in the interstices of the embryo tissues being usually produced by development of some of the interstitial cells; while those which are formed at borders or surfaces appear as usually to be produced by outgrowth from subjacent vessels.* Mr. Hunter, guided by the analogy of the formation of the first bloodvessels in the germinal area of the embryo-chick, was induced to think they must be similarly formed in lymph; and that from its materials both blood and enclosing vessels may be constructed, as it were, in an isolated system, which, at a later period, opens into communication with the vessels and moving blood of the adjacent parts. Rokitansky, and many others, hold the same opinion; while Mr. Travers, Mr. Quekett, and many others, maintain that the lymph forms neither vessels nor blood, but receives those that are projected into it from the parts on or in which it is placed. With this view I fully concur, resting on several grounds:—

1. The direct observations supposed to prove that blood is formed in lymph are very liable to fallacy, through the facility with which blood may be accidentally mixed with the lymph, in consequence of hæmorrhage during life or after death, or in the preparation of the specimens. Where these sources of fallacy have been avoided, I have never seen anything suggestive of a transformation of lymph into blood.

2. The development of blood from tissue-cells is limited, naturally, to the earliest period of embryo-life, as if it needed the greatest amount of force for development; afterwards, blood is not formed except through a long process of elaboration, and with the aid of many organs. Its formation, therefore, in the mal-conditions of inflammation is very improbable.

3. In no specimen of inflammatory lymph have I seen appearances of transitions from lymph-cells to blood-cells, such as we may see in the lymph of the lymphatics, both before and after it is poured into the bloodvessels.

4. Neither in any lymph have I seen appearances of such stellate cells as the interstitial bloodvessels of the early embryo are formed from; nothing comparable with them have ever come into view.

5. In the formation of vessels for granulations and the walls of chronic abscesses, all is favourable to the belief that they grow-up from the bloodvessels of the adjacent parts; and there are no structures to which the lymph bears so close analogy as it does to these, or to which it is so likely to be conformed in the production of its vessels.

On the whole, although direct observations are wanting, I think we may conclude that all the vessels of inflammatory lymph are formed by outgrowth from adjacent vessels. The process we may believe to be similar to that seen in the formation of vessels along the border of the growing tadpole's tail, or at the edges of the healing wound in the frog's web. On certain points of some vessel slight lateral dilatations appear: these extending acquire the form of pouches or diverticula, which, continuing to grow out, at length bend towards each other, meet, and coalesce, so as to form a new vascular loop, an outgrowth from the vessel on which they arose. From such a loop a new one may be formed: and by similar coalescing outgrowths channels may be formed between adjacent vessels, or when two surfaces of lymph or granulations come in contact, their several vessels may be thus by intermediate projections joined. Thus granulations are united in the healing of wounds by secondary adhesion; and thus the adhesions formed in inflammations of serous membranes introduce anastomoses between the visceral and parietal bloodvessels.

In the first instance, the bloodvessels of lymph appear to be usually very numerous and thin-walled; therefore easily bursting, or dilated by congestions during life, or in the attempt to inject them after death. The College-collection contains an extremely beautiful specimen of soft recent lymph from the pericardium of a Cheetah, the vessels of which, injected by Mr. Quekett, appear as numerous and close-set as those of some of the more vascular mucous membranes. They present occasional slight and gradual dilatations, especially when they branch or anastomose. But after an uncertain time, as the lymph becomes more highly organised, so its vessels waste and diminish in number; and while it acquires

* See further, in the Lectures on Repair.

the proper structure of the fibro-cellular tissue, so it descends to the low degree of vascularity of that tissue. The vessels of false membranes are usually rather wide apart, long, slender, and of uniform diameter. In all these particulars they differ from those of more recently vascularised lymph; and their changes are, in these respects, parallel with those of the vessels of granulations during the gradual formation and perfecting of scars.

Perhaps the most perfect instance of the conformity with the natural tissues of the body to which the developed lymph can attain, is manifested in its acquiring a supply of lymphatic vessels. We owe the knowledge of the lymphatics of false membranes to the masterly skill of Professor Schroeder van der Kolk, whose preparations of them are described and represented by his pupil, Dr. de Lespinasse.* Beautiful networks of lymphatics, with their characteristic beaded forms and abundant anastomoses, are shown traversing adhesions extending between two lobes of a lung, and between a liver and the peritoneal covering of the diaphragm; while yet closer networks are seated in the thickened and opaque-white substance of the pleura, or of false membrane covering it, beneath the adhesions.

It seems to be in only the most perfect state, and when bloodvessels have long existed, that lymphatics are formed in false membranes. In recent lymph S. v. d. Kolk has never succeeded in injecting any; and we can only suppose that they are, like the bloodvessels, produced by outgrowth from the lymphatics of the membrane with which they are connected.

The time in which these complete developments of lymph may be accomplished must vary so much, according to the circumstances of the cases of inflammation, that perhaps no reasonable estimate of it can be made. The experiments of Villermé and Dupuytren† upon dogs assign twenty-one days as the earliest time in which new vessels are formed; but I am disposed to agree with Dr. Hodgkin, that a shorter time is sufficient. On the other hand, I am sure that the supposition of their being formed in one or two days is incorrect. The principal case in support of this opinion is that recorded by Sir Everard Home; but the specimens preserved in the College museum‡ show that he was deceived as to the true nature of the case. He says* that he operated for strangulated hernia in a man, and found in the sac a portion of ileum, which was healthy, except in that its vessels were turgid with blood. The patient died twenty-nine hours after the operation; and on examination "several small portions of exuded coagulated lymph" were found adhering to the intestine that had been protruded. When the vessels of the intestine were injected, the injection passed into vessels in all these portions of supposed lymph, each "having a considerable artery.....and a returning vein." Sir Everard Home, therefore, concludes "that the whole operation of throwing out coagulable lymph, and supplying it with bloodvessels after it had become solid, was effected in less than twenty-four hours."

Now, one of these specimens was figured by Mr. Hunter,† "to show a small portion of coagulating lymph.....which is supplied with vessels;" but neither here, nor in his manuscript catalogue, does he allude to a probability of the vessels having been formed in twenty-four hours, although, had he believed it, he would scarcely have failed to record it.‡ An examination of the specimens shows that the small shred-like portions of membrane, attached by little pedicles to the intestine, have not the appearance of recently coagulated lymph, but are fully organised, with traces of filaments and fat-cells. They are also very regularly disposed, at distances of from half an inch to an inch from each other, and are nearly all placed in two rows on each side of the intestine, about half an inch from the attachment of the mesentery, like very minute appendices epiploicæ, such as are occasionally met with on the coats of the small intestine. Whether they be such appendices or not, it is in the highest degree improbable that they were formed after the operation; especially since they are too minute and delicate to have prevented the intestine from exhibiting, when exposed in the sac, the natural polished appearance of its surface.

I am not aware of any other case adapted to prove the earliest period at which bloodvessels may be formed in lymph. Serous surfaces may, indeed, become adherent in twenty-four hours, but this does not imply vascularity of the lymph between them; it is simply adhesion by the coaptation of the intermediate lymph.

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* Spec. Anat. Path. de Vasis novis Pseudomembranarum, 8vo. Daventriæ, 1842, figs. iii. iv.

† Quoted by Dr. Hodgkin in his Lectures on the Morbid Anatomy of the Serous Membranes, p. 39.

‡ Nos. 81 and 82 in the Pathological Museum.

* In his Dissertation on Pus, p. 41. The whole case is given in the College Catalogue, vol. i. p. 37.

† On the Blood, pl. vi. fig. 2.

§ In the same work (p. 350) he speaks of nine days as a short time for the complete organisation and adaptation of adhesions.

LECTURE IV.

Degenerations and diseases of the lymph-products of inflammation.—General account of degenerations, as normal changes to which all living parts are prone; their relations to the other modes of natural nutrition, and their purposes; their difference from diseases.—Varieties of normal degenerations.

Characters of these degenerations when they affect inflammatory products in their several stages of development.—Degenerations of fibrine; its withering, liquefaction, disintegration, fatty, calcareous, and pigmental degenerations. — Degenerations of lymph-corpuscles; their withering, degeneration into granule-cells, and disintegration; their calcareous and pigmental degenerations: formation of various pus-cells, and their subsequent degenerations, as observed in the contents of chronic abscesses—Degenerations of adhesions, and other examples of fully organised lymph—Diseases of the products of inflammation.

In the last lecture a general history was given of the chief developments of the lymph exuded in the inflammatory process. I propose now to tell a corresponding history of its degenerations; and herein to describe what appear to be the transitions from the ordinary forms of lymph in its primary state,—its fibrine and its corpuscles,—to those many lower forms enumerated as molecular and granular matter, as pus-cells, granule-cells, inflammatory globules, and the rest. I said that, for the development of lymph produced in inflammation, it is requisite that the inflammation shall have ceased, and the conditions of healthy nutrition be restored. In the failure of this event, if the inflammation continue, or the due conditions of nutrition be in any way suspended, then, instead of development, degeneration may ensue. And this may happen in any of the stages of formation which I described in the last lecture: it may happen alike to the rudimental fibrine, or to the earliest lymph-cell, or to either, in any part of its progress to complete development.

Allow me first to illustrate generally what is to be understood by degeneration, as distinguished from disease, and to point out some of the larger principles which the very active inquiries of late years made enable us to enunciate concerning degenerations.

The usually admitted modes of normal operation of the nutritive processes are three—namely, development, growth, and maintenance or assimilation.* The accepted meanings of these terms are known; and I need only say concerning them that the maintenance of a part by assimilation must not imply the maintenance of an unchanged state, but a series of minor progressive changes slowly worked in the part, and consistent with that exercise of its functions which is most appropriate to the successive periods of its existence.

But to these three we should add, as another normal method of nutrition, the process of natural degeneration, such as occurs in advanced life. To degenerate and die is as normal as to be developed and live: the expansion of growth, and the full strength of manhood, are not more natural than the decay and feebleness of a timely old age,—not more natural, because not more in accordance with constant laws, as observed in ordinary conditions. As the development of the whole being, and of every element of its tissues, is according to certain laws, so is the whole process regulated, by which all that has life will, as of its own workings, cease to live. The definition of life that Bichat gave is, in this view, as untrue as it is illogical. Life is so far from being "the sum of the functions that resist death," that it is a constant part of the history of life that its exercise leads naturally to decay, and through decay to death.

Of the manner in which this decay or degeneration of organisms ensues we know but little. Till within the last few years the subject of degenerations was scarcely pursued: and, even of late, the inquiries, which ought to range over the whole field of living nature, have been almost exclusively limited to the human body. The study of development has always had precedence in the choice of all the best workers in physiological science. They who have devoted many years of laborious

* Lectures on Nutrition, in Medical Gazette for 1847.

thought and observation to the study of the changes by which the living being is developed from rudiment to perfection, have given fewer hours to the investigation of those by which, from that perfection, it naturally descends into decay and death. Almost the only essays at a general illustration of the subject have issued in the ridiculous notion that, as the body grows old, so it retrogrades into a lower station in the scale of animal creation. The flattened cornea is supposed to degrade the old man to the level of the fish; while the *arcus senilis*, by a fancied correspondence with an osseous sclerotic ring, maintains him in the eminence of a bird; his dry thick cuticle makes him like the pachydermata; and his shrivelled spleen approximates him to the humility of the mollusk. One can only commend such day-dreams to the modern supporters of the doctrine of transmutation of species; and they might, indeed, form an appropriate supplement to their scheme, if they would maintain that, in these latter days, our species is destined to degenerate into lower and yet lower forms, descending through the grades by which, in by-gone times, it ascended towards its climax in humanity.

One cannot but wonder at the comparative neglect with which wiser men than these philosophers have treated a study so full at once of interest and of importance as this of the natural degeneration of the body. It could not be without interest to watch the changes of the body as life naturally ebbs,—changes by which all is undone that the creative force in development had achieved,—by which all that had been gathered from the inorganic world, impressed with life, and fashioned to organic form, is restored to the masses of dead matter,—to trace how life gives back to death the elements on which it had subsisted,—the progress of that decay through which, as by a common path, the brutes pass to their annihilation, and man to immortality. Without a knowledge of these things our science of life is very partial, very incomplete. And the study of them would not lack that peculiar interest which appertains to inquiries into final causes; for all the changes of natural decay, or degeneration, in living beings, indicate this purpose and design—that, being gradual approximations to the inorganic state of matter, they lead to conditions in which the elements of the body, instead of being on a sudden and with violence dispersed, may be collected into those lower combinations in which they may best rejoin the inorganic world; they are such, that each creature may be said to die through that series of changes which may best suit it, after death, to discharge its share in the economy of the world, either by supplying nutriment to other organisms, or by taking its right part in the adjustment of the balance always held between the organic and the inorganic masses.

Nor would the student of the design of these degenerations do well to omit all thought of their adaptation, in our own case, to the highest purposes of our existence. When, in the progress of the "calm decay" of age, the outward senses, and all the faculties to which they minister, grow dim and faint, it may be on purpose that the Spirit may be invigorated and undisturbed in the contemplation of the brightening future; that, with daily renewed strength, it may free itself from the encumbrance of all sensuous things, or retain only those fragments of thought or intellectual knowledge which, though gathered here on earth, yet bear the marks of truth, and, being Truth, may mingle with the truth from Heaven, and form part of those things in which Spirits of infinite purity and knowledge may be exercised.

Moreover, the changes of natural degeneration in advanced life have a direct importance in all pathology, because the right understanding of them may guide us to the interpretation of many anomalies which, while they occur in earlier life, we are apt to call diseases, but which are only premature degenerations, and are to be considered, therefore, as defects, rather than as perversions, of the nutritive process, or as diseases only in consideration of the time of their occurrence.*

In this view, it will be a great help to our knowledge of disease if we can determine and arrange all the changes that mark the progress of natural decay or degeneration, *i. e.* the decay of old age, or of naturally abrogated functions. They seem to be these—1. Wasting or withering; the latter term may imply the usually coincident wasting and drying of a tissue. 2. Fatty degeneration, including many of what have been called granular degenerations. 3. Earthy degeneration, or calcification. 4. Pigmental degeneration. 5. Thickening of primary membranes.

Of each of these let me cite one or two examples.

Of *withering*, or wasting and drying, which is perhaps the commonest form of

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* One can here have in view only the cases in which the degeneration affects the whole, or some considerable part of an organ; for it is very probable that some of the degenerations which we see *en masse* in the organs of the old, or in the seats of premature defect of nutrition, are the same as occur naturally in the elementary structures of parts, previous to their being absorbed and replaced, as it were by one particle at a time, in the regular process of nutrition.

atrophy, we have abundant instances in the emaciation of old age; in which, while some parts are removed by complete absorption, others are only decreased in size, and lose the succulency of earlier age.

Of the *fatty degeneration* in senility, we have the best proofs, as to its general occurrence, in the increasing obesity which some present at the onset of old age, and in the general fact that there is more fatty matter in all the tissues, and most evidently in the bones, than there is in earlier life; while, for local senile fatty degeneration, we find the *arcus senilis*, or fatty degeneration of the cornea, according to the interesting discovery by Mr. Canton,* and the accumulating fatty or atheromatous degenerations of arteries.

The *calcareous degeneration* is, in old age, displayed in the gradually increasing proportion of earthy matter in the bones; in the extension of ossification to cartilages, which, in all the period of vigour, had retained their embryonic state; and in the increasing tendency to earthy deposits in the arteries, and other parts.

The *pigmental degeneration* has its best instances in the gradually accumulating black pigment spotting and streaking the lungs; in the slate or ash-colour which is commonly seen in the thin mucous membranes of the stomach and intestines of old persons;† and in the black spotting of the arteries of some animals, in which pigment seems to hold the place of the fatty degenerations so usual in our own arteries.‡

Of the *thickening of primary membranes* we have indications in the usual thickening of the tubules of the testes, and, I think, of some other glands, as their function diminishes in old age; in the opaque white thickening of the primary or inner membrane of nearly all bloodvessels; and in the thickening of the walls of cartilage-cells in some forms of ossification. To this, also, we have a strong analogy in the thickening of the cell-walls of the heart-wood of plants.

Such are the changes which, singly or in various combinations, constitute the most evident degenerations of old age in man. Their combinations give rise to numerous varieties in their appearance, such as, *e. g.*, the increase of both fatty and earthy matter in old bones; the fatty degeneration and wasting of the pulmonary membrane in the emphysematous lung; the dry, withered, and darkly tinged epidermis; the coincident fatty and calcareous deposits in old arteries. But I need not dwell on these, nor on the conditions which determine the occurrence of one rather than another mode of degeneration; for these I cannot tell. Let me rather observe that, taken singly, all the changes I have enumerated display certain characters, which should be present in every process that may be justly called a degeneration, and distinguished from disease. In all true degenerations these, or most of these, following characters should concur:—

First, and before all others, it should be a change naturally and usually occurring, in one or more parts of the body, at the approach of the natural termination of life, or, if not then beginning, yet then regularly increasing.

2. It should be a change in which the new material is of lower chemical composition, *i. e.*, is less remote from inorganic matter than that of which it takes the place. Thus, *e. g.*, fat is lower than any nitrogenous organic compound, and gelatine lower than albumen, and earthy matter lower than all these.

3. In structure, the form should be less developed than that of which it takes the place: it should be either more like inorganic matter, or less advanced beyond the form of the mere granule or the simplest cell. Thus the approach to crystalline form in the earthy matter of bones, and the crystals in certain old vegetable cells, are characteristic of degeneration; and so are the granules of pigment and of many granular degenerations, and the globules of oil that may replace muscular fibres or the contents of gland-cells.

4. In function, the part should have less power in its degenerate than in its natural state.

5. In its nutrition, it should be the seat of less frequent and less active change, and without capacity of growth, or of development.

Such are the characters of degenerations; and there are many things which show that the assumption of these characters is to be ascribed to a defect, not to a perversion, of the vital force, or of the conditions of nutrition.

Thus (*a*) these are all apt to occur in a part of which the functions are abrogated: a motionless limb wastes or becomes fatty as surely as an old one does. (*b*) They often occur, too, in parts that fail to attain the development for which they seemed to be intended. Thus, *e.g.*, fatty degeneration usually ensues in the cells of unfruitful Graafian vesicles.* (*c*) They bear also a certain

* Lancet, April 1850.

† It is remarkable that this accumulation of pigment should be occurring in any set of organs while pigment is disappearing from other parts; such as the hair.

‡ I have seen a similar black pigmental degeneration of the minute arteries of the human brain.

* Reinhardt in Traube's Beiträge, B. i.

general analogy to the changes that ensue in some of the materials that are habitually excreted from the body; in the construction or composition of which materials one seems to have an instance of the gradual supervention of the ordinary or imitable processes of chemistry. (*d*) Again, they display living parts tolerating the presence and incorporation of inorganic or dead matter; fat being commonly infiltrated about degenerate cells and membranes, and earthy matter with it, even in the crystalline form. (*e*) And, lastly, and perhaps most clearly, the origin of degenerations from defective, rather than from perverted, conditions of nutrition, appears in the fact that one, at least, of them may be produced artificially. The fact observed by Dr. Quain,* that the fatty degeneration of muscular fibre is closely imitated when healthy dead muscle is placed in dilute acid, suggests, as the formation of adipocire did to Dr. Williams† that, during life, the change of the protein-compound, which leaves the residuum of fatty matter, is one of common, that is, of imitable chemistry. Now we may be sure that where this chemistry prevails, there the forces or conditions of life are in defect.

From the whole, we may safely hold that, as the changes to which the several tissues are naturally prone in old age are the results of defect, not of perversion, of the nutritive process, so are the corresponding changes when they happen in earlier life; although, through their appearing prematurely, they may in this case bear the features of disease. Therefore, we should be prepared to separate from the catalogue of diseases all those changes in which we find the marks of degeneration alone; for the distinction of degeneration from diseases must be an essential, though often it may be an obscure, one. The one is natural, though it may be premature; the other is always unnatural: the one has its origin within, the other without, the body: the one is constant, the other as various as the external conditions in which it may arise: finally, to the one we are prone, to the other only liable.

I will now proceed to describe what, in accordance with the foregoing observations, appear to be the chief degenerations of inflammatory products. As I have said, the degeneration may begin in any period of the formation of the lymph, as well in its rudimental as in its fully organised state; and that which determines for degeneration rather than development seems always to be the continued suspension of the conditions of nutrition.

The following appear to be the chief degenerations of the fibrine of lymph, or of the materials derived from its earliest stages of development:—

1. It may wither—wasting, and becoming firmer and drier, passing into a state which Rokitansky* has designated *horny*. One sees the best examples of this change of fibrine in the vegetations on the valves of the heart, or in the large arteries, when they become yellow, stiff, elastic, and nearly transparent.† The fibrine may, in this state, show no marks of development into tissue, but may have all the simplicity of structure of ordinary fibrine, being only drier and more compact. A similar character is nearly acquired when lymph is deposited over a lung which is extremely compressed in empyema, or in hydrothorax.‡ The tough dry lymph that here forms the greyish layer over all the lung is not always developed, though it may adhere firmly: it is withered, wasted, and dried (as the lung itself may be) in consequence of the compression.

2. Fibrine is subject to a degeneration which we may compare with fatty degeneration. In the coagula, or solid parts of effusions, that are found in the lower forms of inflammation, or in very unhealthy persons, the fibrine is usually not clear and uniform, and filamentous, but rather opaque or turbid, nebulous or dotted, presenting just such an appearance as marks the earliest stages of fatty degenerations in the muscular fibrils. In such coagula as these, also, one sees, not unfrequently, minute, shining, black-edged particles, which we may know to be drops of oil; while some general alteration in the composition of the fibrine is shown by its not being made transparent with acetic acid. In all such cases as these the fibrine is also very soft, and easily broken: it is devoid of all that toughness and elasticity which is the peculiar characteristic of well-formed fibrine; and by breaking it up, one may see the meaning of what one so often finds in the lowest forms of inflammatory exudation, such as occur in erysipelas and typhus,—namely, films and fragments of molecular and dotted substance, floating in fluid that is made turbid by them, and by abundant

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* Lancet, Report of Medico-Chir. Soc., April, 1850.

† Principles of Medicine. Rokitansky has always held that the fatty and earthy matters of degenerations are residues of the transformations which the higher compounds of the tissues have undergone; part of the products of the transformation being absorbed, and part (at least in many cases) remaining in the place. See, also, the Lectures on Nutrition, p. 40.

* Vol. i. p. 229.

† As in a case in the Museum of St. Bartholomew's, ser. xii. 55.

‡ As in the College Museum, No. 1756 A, and 1757.

minute molecules and granules and particles of oily matter. These represent the disintegration of fibrine that has degenerated after clotting, or has thus solidified in an imperfect coagulation. Of such changes, also, an excellent instance is presented in the softening and disintegration of the clots within the heart, which Mr. Gulliver* has described, and which might be studied for the illustration of the corresponding changes in inflammatory lymph.

We have examples of numerous varieties of this degeneration and disintegration of fibrine formed in inflammation. It is a principal constituent of most of what has been called "aplastic lymph," in inflammation of the serous membranes. Similar fibrine occurs, mingled with mucus, in the severer inflammations of the mucous membranes. And to the same source we may trace most of that molecular and granular matter which is usually mingled with pus formed by the suppuration of inflammatory indurations—with the variously changed corpuscles of "scrofulous matter" — or with the granule-cells, and other corpuscles of pneumonia, and the like inflammations. At least, this disintegration of fibrine is probably a frequent origin of such molecular matter; while the quantity of fatty matter present in pus and the products of pneumonia, and its gradual increase,† while pus is retained in an abscess, confirm the view, that the changes here described are of the nature of fatty degeneration.

When we see how a large mass of inflamed hard substance will become fluid, as it suppurates, and this with scarcely any, if any, increase of bulk, we may believe that another change of which fibrine is capable in degeneration is its liquefaction. In such a swelling as a hard bubo, or a hard chronic abscess, we may be nearly sure there is coagulated fibrine, both from the general circumstances of the inflammation, and because neither corpuscles alone, nor fibrine in the liquid state, would give such hardness. The suppuration, therefore, of such a swelling, if without increase of bulk, can hardly be explained, except on condition of the fibrine, which had coagulated, becoming again liquid. The occasional liquefaction of clots out of the body‡ makes this more probable; but I am not sure that it can be proved by any more direct facts.

I may here refer to a point of some practical importance connected with these forms of degeneration of lymph, whether affecting fibrine or corpuscles. When the fibrine has withered and become dry, it is probably put out of the capacity of being further developed, and is rendered passive for further harm or good, except by its mechanical effects. But the molecular and fatty degenerations are yet more beneficial, in that they bring the lymph into a state favourable to its absorption, and, therefore, favourable to that which is termed the "resolution" of an inflammation in which lymph has been already formed. I suppose it may be considered as a general truth, that the elements of a tissue cannot be absorbed so long as they retain their healthy state. There is no power of any absorbent vessels that can disintegrate or decompose a healthy portion of the body: for absorption, there must be not only an absorbing power, but also a previous or concurrent change, as it were a consent, in the part to be absorbed; so that it may be reduced (or, rather, may reduce itself) into minutest particles, or may be dissolved. And this change is probably one of degeneration, not death, in the part; for dead matter is rather discharged from the body than absorbed.

Now there are some facts which indicate the probability that the fatty degeneration is that which commonly precedes the natural absorption of many normal parts; or, rather, that, in the change which they undergo before absorption, fatty matter is one of the products, and that the principal evident difference between the atrophy of a part which is manifested by its wasting, and the atrophy which is manifested by fatty degeneration, is, that the fatty matter, which is absorbed in the former case, is retained in the latter. However this may be, it is certain that the disintegration and fatty degeneration of the fibrine-products of inflammation bring them into a state most favourable for absorption; indeed, one may see in fibrine thus changed many things which, in regard to the fitness for absorption, make it parallel with chyle.* Of such absorption of fibrine we may find many instances. In rheumatic iritis we may believe the lymph to be fibrinous; but we see its complete absorption taking place; and the recent observations of Dr. Kirkes on the rarity of adhesions of the

* Medico-Chirurgical Transactions, vol. xxii.

† Gerber, Allgem. Anatomie, 48.

‡ As in cases by Nasse and De la Harpe, quoted by Henle, in his Zeitschrift, B. ii. p. 169. See also Virchow on the same subject, in the Zeitschrift, B. iv. Henle refers to this same liquefaction the changes that ensue in emitted semen.

* See also the ingenious contrast of the progress of chyle and the regress of pus drawn by Gerber, in his Allgem. Anatomie, p. 49.

One assumes that the absorption of visible particles is possible here, as it is commonly assumed to be in chyle; but the passage of such particles through the membranous walls of blood-vessels is only imaginable. Can it be that they pass into the vessels by being incorporated in the tissue adjacent to them, and then by being gradually shifted into them in the mutations of particles that are effected in nutrition?

pericardium, in comparison with the frequency of percarditis,* may be in the same manner explained. In rheumatic pericarditis we may be sure fibrine is effused; and the observed friction-sound has, in at least one case, proved its coagulation; yet in this case, when death occurred four months afterwards, scarce a trace of fibrine was found in the pericardium: it had been absorbed, and the degeneration I have been describing was probably the preparation for its absorption.

3. I am not aware of any direct proof of the calcareous degeneration ensuing in the fibrinous part of an inflammatory exudation; but we have the strongest evidence from analogy for believing that this change may be a frequent one. For there are numerous instances of calcifications of fibrine within the vessels—as, *e. g.* in the ordinary formation of phlebolithes from clots of blood, in the branching and irregular pieces of bone-like substance found in obliterated veins, and in the lumps and grains of substance like mortar imbedded in fibrinous deposit in the heart's valves. We can, therefore, hardly doubt that the fibrine, even before development, may take part in formations of earthy matter in inflammatory products; but the calcareous degeneration is much more frequent in purulent fluids, and in the later developments of lymph.

4. Lastly, we have examples of the pigmental degenerations of fibrinous lymph in the various shades of grey and black which often pervade the lymph formed in peritonitis, and which are produced, not by staining or discoloration of the blood by intestinal gases, but, according to Rokitansky, by the incorporation of free pigment-granules.

Such appear to be the degenerations of the fibrinous lymph of inflammation: such at least are the changes in it which we may refer to defects in its power or conditions of nutrition, because they correspond with changes that may be traced in the gradual degenerations of old age. I need hardly say, that it is only by such correspondence that we can interpret them; for when we find them, it is altogether beyond our power to tell, by direct observation, whether, or in what way, the conditions of nutrition were defective.

The corpuscular constituents of lymph, in any of their stages of development, may retrograde, and then present degenerations corresponding, and often concurrent, with those which I have just described.

a. Their withering is well seen in some forms of what is called scrofulous matter, such as occur in chronic and nearly stationary scrofulous enlargements of lymphatic glands. In the dull ochre-yellow-coloured and half dry material imbedded in such glands, may be found abundant cells, collapsed, shrivelled, wrinkled, glistening, and altogether irregular in size and form. One might suppose them to be the remnants of pus dried-up, or the corpuscles of chronic tuberculous matter, if it were not that among them are some with nuclei shrivelled like themselves, and some elongated and attenuated, which are evidently such as withered after they had been developed into the form of fibro-cells; into which form it is certain that neither pus-cells nor tubercle-cells are ever changed.

These are the best examples of withered lymph-corpuscles; but they may be also found in the pus of chronic abscesses, especially, of course, in that of such abscesses as ensue by suppuration of lymph-deposits like those just referred to. It may be hard sometimes to say whether corpuscles in these cases may not be pus-corpuscles shrivelled up: but, on the whole, I am inclined to believe that the shrivelled corpuscles of the pus of chronic abscesses are usually derived from the lymph, in which, having withered, they had become incapable of further change.

b. The fatty degeneration of lymph-cells is shown in their transition into granule cells.* We owe the first demonstration of this to the excellent observations of Reinhardt,† who has also shown how, by similar degenerations, corresponding forms of granule-cells may be derived from the primary cells of almost all other, both normal and abnormal, structures.

This method of degeneration appears peculiarly apt to occur in the inflammations of certain organs—as, especially, the lungs,‡ brain, and spinal cord; but it may be found occasionally prevalent in the lymph of nearly all other parts, and in the granulations forming the walls of abscesses or of fistulæ. It may occur alike in the early forms of lymph-cells, and after they have already elongated and attenuated themselves, as for the formation of filaments, and after they have degenerated into pus-cells. The changes of transition are briefly these:—The lymph-cells, which may have at first quite normal characters, such as I have assigned to "pri-

* Medical Gazette, April 1849.

* The exudation-cells of Dr. Hughes Bennett; the inflammatory globules of Gluge.

† Traube's Beiträge, B. ii. 217, e. s.

Observations similar to part of those of Reinhardt were made independently by Dr. Andrew Clark (see Medical Gazette, vols. xlii. xliii.)

‡ Dr. Gairdner describes also the formation of granule-cells from epithelium-cells in pneumonia (Contributions to the Pathology of the Kidney, p. 20).

mordial cells," present a gradual increase of shining black-edged particles, like minute oil-drops, which accumulate in the cell-cavity, and increase in number, and sometimes in size also, till they nearly fill it. The fatty nature of these particles is proved by their solubility in ether: and their accumulation is attended with a gradual enlargement of the cell, which also usually assumes a more oval form. Moreover, while the fatty matter accumulates, the rest of the contents of the cell becomes very clear, so that all the interspaces between the particles are quite transparent; and, coincidently with all these changes, the nucleus, if any had been formed, gradually fades and disappears.

I need hardly say, that in these particulars the changes of the lymph-cells, (which may also occur when they have been already developed into the form of fibro-cells,) correspond exactly with those of the fatty degenerations observed in the cells of the liver or kidney, or in the fibres of the heart. There can be hardly a doubt of the nature of this process; and it presents an important parallel with the similar changes described in fibrine. For, we may observe, first, that where this degeneration is apt to occur in lymph, it is least likely to be developed. A proper induration and toughening of the lungs and brain, such as might happen through development of the products of inflammation, is extremely rare; it is scarcely seen, except in the scars by which the damages of disease are healed. And, besides, this degeneration is a step towards the absorption of the lymph; for commonly we may trace yet later stages of degeneration in these granule-cells. They lose their cell-walls, and become mere masses of granules or fatty particles, held together for a time by some pellucid substance, but at last breaking up, and scattering their components in little clusters, or in separate granules.

Thus, if at no earlier period of their existence, or after no fewer changes, the lymph-corpuscles may pass into a condition as favourable for absorption as is that of the fibrine when similarly degenerate and broken up: and such as this, we may believe, is a part of the process by which is accomplished that "clearing up" of the solidified lung, which is watched with so much interest in pneumonia.

c. I may speak very briefly of the calcareous and the pigmentary degenerations of the lymph-cells. The former appear in cases such as Henle* refers to, in which granule-cells are composed not wholly of fatty matter, but in part also of granules of earthy matter. In this combination they correspond with the common rule; for the fatty and earthy degenerations are usually coincident: they are combined in the advanced stages of the degenerations of arteries, and may be said to have their normal coincidence in ossification. Of the pigmentary degeneration of lymph-cells there are, I suppose, examples in the black matter effused in peritonitis: but the best examples are in the cells of bronchial mucus, to which I have already referred.

But I pass quickly by these, that I may speak of the degenerations of lymph-cells into pus-cells, or, more generally, of the process of suppuration that is connected with inflammation. The purulent is the most frequent degeneration of lymph, ensuing in nearly all cases in which it is placed in conditions unfavourable to its development—as, in the persistence of inflammation, or in exposure to air, or in general defects of vital force.

Of the conversion of lymph into pus we have numerous evidences; but a few may suffice for the proof.

1. The fluid of such vesicles as those of herpes is, in the first instance, a pure lymph, containing corpuscles which might be taken as types of the lymph- or exudation-corpuscles. If we watch these vesicles, we see their contents not increased,—rather, by evaporation, they are diminished; but the lymph is converted into pus, and pus-cells are now where lymph-cells were. And the changes may ensue very quickly: I think I have known it accomplished in twelve hours at the most.

2. In like manner, when we watch the progress of an abscess, we may find one day a circumscribed, hard, and quite solid mass, and in a few days later the solid mass is fluid, and this with little or no increase of bulk. Now the incipient solidity and hardness are due to lymph; the later fluid is pus, and the change is the conversion of lymph into pus.

3. The relation of lymph- and pus-cells is also shown in this; that they often cannot be distinguished from one another. Even in common suppuration of a granulating wound, the granulation-cells (which cannot be distinguished from lymph-cells) appear to be convertible into pus-cells; superficial cells being detached in pus, while deeper ones are being developed into filaments. But in worse-formed granulations, as in those lining a sinus or fistula, the cells are often by no characters, except by their forming a solid tissue, distinguishable from pus-cells. One may see the same conversion of lymph into pus thus illustrated: an amputation through the thigh was performed when all the parts divided were infiltrated with lymph effused in connec-

* In his Zeitschrift, B. ii.

tion with acute traumatic inflammation of the knee-joint. Next day pus flowed freely from the wound. Now, in an amputation through healthy tissues, free suppuration does not appear till after three or four days: the pus here seen must have been formed by the conversion of the lymph previously infiltrated in the divided tissues.

These facts, while they may prove that the pus-cells are commonly the result of degeneration of lymph-cells, may also serve to show that the question whether pus-cells are ever formed in another way, or are ever an original or primary product of inflammation, cannot be positively settled. We cannot always discern a preliminary lymph-stage; but neither can we always distinguish lymph-cells from pus-cells, nor can we see in how very brief a time the transformation may be accomplished.

Assuming, however, that, when it appears as an inflammatory product, the pus-cell is always either an ill-formed or a degenerate lymph-cell, we may believe that, of the various forms which it presents, some depend on the peculiar defectiveness of the lymph; some on the state which the lymph-cell, previously well-formed, had attained before its degeneration began; and others on the further degenerations which may have taken place after the characters of the pus-cell have been acquired.*

I would especially observe, that in specimens of what might be called "good" pus, we may find three principal forms. There are—1st, some corpuscles presenting the peculiar and well-known granulated or wrinkled appearance of pus-cells, but from which water will raise up no cell-wall; 2d, from others, like these at first sight, water will raise a cell-wall, and will show that the former kind consist of only such a substance as forms the contents of these; 3d, in others, even when no water is added, a cell-wall is visible, and within this are granulated contents, with a more or less distinct nucleus imbedded in them. In all these forms, moreover, the addition of acetic acid usually displays a single or a bipartite or tripartite nucleus. Now, it may be that these represent three different stages of the pus-cell, either developing, or, more probably, degenerating; but I think it is much more likely that these forms are the results of the purulent degeneration beginning in lymph-cells at different stages of their development. There is so remarkable a correspondence between these three varieties of pus-corpuscles, and the three chief forms which I described as observable in the development of the primordial cell of lymph, that one cannot but suspect that the three forms in the pus represent corresponding and similar degenerations from the three forms in the lymph.

When once formed, the pus-cells, if they are retained within the body, have no course but to degenerate further; it is characteristic of their being already degenerate, that they can neither increase nor develope themselves. The various corpuscles found in pus, besides those I have already mentioned, must find their interpretation in these degenerations; for the pus-cells are prone to all the degenerations that I described as occurring in the lymph-cells.

a. They may wither, as in the scabbing of pustular eruptions, or in long-retained and half-dried strumous abscesses.

b. Or, they may be broken up, whether before or after passing into the fatty degeneration, which is one of their most common changes, and in which they are transformed into granule-cells. It is this breaking-up into minute particles which, probably, precedes the final absorption of pus.

c. Or, lastly, both the cells and the fluid part of the pus may alike yield fatty and calcareous matter, and this may either remain diffused in fluid, or may dry into a firm mortar-like substance.

It is to such degenerations as these, in various degrees and combinations, and variously modified by circumstances, that we must ascribe the diverse appearances of the contents of chronic abscesses and of the substances left after their healing. In such abscesses we may find mixtures of pus-cells, granule-cells, and molecular matter, diffused in more or less liquid; or all cells may be broken up, and their debris may be found mingled with minute oily particles, which appear in all such cases to be always increasing; or, with these may be abundant crystals of cholesterine, or such crystals may predominate over all other solid contents. In yet other chronic abscesses (though, still, without our being able to tell why the pus should degenerate in these rather than in the foregoing methods), we find molecules of carbonate and phosphate of lime, mixed with fat-molecules and crystals, which are diffused in an opaque white fluid, and look like the deposit of lime-water, or like white paint; and as these contents dry, in the healing of the abscess, so are formed the mortar-like deposits and the hard concretions, such as are found in the substance of lymphatic glands, or other organs that have been the seats of chronic abscesses.

Time and patience would fail in an attempt to describe all the varieties of mate-

* It may be added that other rudimental cells, besides those of lymph, may be so altered as to resemble exactly the ordinary pus-cells. In many of the supposed cases of pus in the blood, the bodies taken for pus-cells were certainly only altered white blood-corpuscles.

rial that may thus issue from the transformations of pus. What I have enumerated are the principal or typical forms with which I believe nearly all others may be classed.

In conclusion of this part of the subject, a few words must be added respecting the degenerations which may occur after the lymph-products of inflammation are completely organized. All these degenerations, to which I have now so often referred, may be observed in fully-formed adhesions, or in the corresponding organized tissues in the substance of organs.

Of the wasting of adhesions we often see instances in the pericardium, where films of false membrane are attached to one layer of the membrane, while the opposed portion of the other layer is only thickened and opaque. A more remarkable instance, also, is presented in a case by Bichat, in which a man made twelve or fifteen attempts at suicide, at distant periods, by stabbing his abdomen. In the situations of the more recent wounds the intestines adhered to the walls of the abdomen; in those of the older wounds, the older adhesions were reduced to narrow bands, or were divided and hung in shreds.

Of fatty degeneration I have seen no good examples in adhesions or similar inflammatory products, but of calcareous degenerations, or of such as present a combination of fatty and earthy matter, museums present abundant specimens. Among these are most of the plates of bone-like substances imbedded in adhesions of the pleura, in thickened and opaque portions of the cardiac pericardium, in the tunica vaginalis, in old hydroceles, in the thickened and nodulated capsule of the spleen, in the similarly altered mitral and aortic valves. So, too, many of the so-called ossifications of muscles and ligaments are examples of calcareous degeneration of fibrous tissue formed in consequence of inflammation of these parts, and imbedded in masses of fibrous-looking bands within their substance. In some of these cases, indeed, there may be an approximation to the characters of true bone; but in nearly all, the earthy matter is deposited in an amorphous form, and seems to take the place of the former substance, as if, according to Rokitansky, it was a residue of the transformation of the more organized tissue, whose soluble parts have been, after decomposition, absorbed.*

Pigmental degeneration of adhesions may be seen, sometimes, in those of the pleura, in which black spots appear like the pigment-marks of the lungs and bronchial glands.* Adhesions of the iris, also, may become quite black; I suppose by the formation of pigment like the uvea.

Such appear to be the chief instances of degeneration ensuing in the more or less developed products of inflammation. Doubtless, the account which I have given of them is very incomplete, and in its details sometimes erroneous; yet I venture to think it may be useful to my fellow-workers in pathology, by helping them to keep the right direction in our labours.

Among the signs of the attainment of complete membership in the economy we may enumerate this,—that the organised product of inflammation is liable to the same diseases as the parts among which it is placed; that it reacts like them under irritation; is like them affected by morbid materials conveyed to it in the blood; and like them may be the seat of the growth of new and morbid organisms. No more complete proof of correspondence with the rest of the body could be afforded than this fact presents; for it shows that a morbid material in the blood, minute as is the test which it applies, finds in the product of inflammation the same qualities as in the older tissue to which it has peculiar affinity.

The subject, however, of the particular diseases to which these substances, themselves the products of disease, are liable, has been little studied. As I have already said, lymph, while it is being highly organised, is often the seat of hæmorrhage; its delicate new-formed vessels bursting under some external violence, or some increased pressure, and shedding blood. Such are most of the instances of hæmorrhagic pericarditis, and other hæmorrhages into inflamed serous sacs.

Even more frequently, the lymph, when organised, becomes itself the seat of fresh inflammation. Thus, in the serous membranes, we may find adhesions in the substance or interstices of which recent lymph or pus is deposited; or, in other cases, adhesions, or the thickenings and opacities of parts, become highly vascular and swollen. It is, indeed, very probable that, in many of the instances of the recurring inflammations that we watch in joints, or bones, or other parts, the seat of the disease is, after the first attack, as much in the organised product of the former disease as in the original tissue.

As an example of the inflammation of organised inflammatory products, I may present a specimen which has

* Numerous specimens of the calcareous degeneration of adhesions were shown from the College Museum, including Nos. 103, 1493, 1494, 1516, 1548, 3367, 3456, &c.

* As in No. 96 in the College Museum.

some historic interest.* It is one of those by which attention was first drawn to the connection between acute rheumatism and disease of the heart. It was presented to the College by Sir David Dundas, in 1808, and he sent with it a letter,† in which he says that it exemplifies a disease of the organ "which is not described by any author that I am acquainted with, but which, from the number of cases which have fallen under my observation, is, I apprehend, very frequent in this country. The most remarkable circumstance of this disease is its being always connected with, or subsequent to, an attack of acute rheumatism." In relation to the subject of which I was speaking, the specimen has this point of further interest, that it shows such a deposit of recent lymph among the old adhesions as, we may be sure, was derived from their vessels.

I suppose, also, that to such inflammations of organised inflammatory products we may ascribe many of the occasional aggravations of chronic inflammations in organs—the renewed pains and swellings of anchylosed joints, of syphilitic nodes, and the like,—which are so apt to occur on exposure to cold, or in any other otherwise trivial disturbance of the economy. In such cases we may believe that the former seat of disease is again inflamed, and that with it are involved the organised products of its former inflammations. And in such cases there are, perhaps, none of the effects of inflammation which may not ensue in the newly organised parts: evidently, they may be softened, or thickened and indurated, and made more firmly adherent: or they may be involved in ulceration, or may slough with the older tissues among which they are placed.

Lastly, the products of inflammation may be the seats of the morbid deposits of specific diseases. In their rudimental state they may incorporate the specific virus of inoculable diseases, such as primary syphilis, variola, and the rest; and when fully organised they may be the seat of cancer and tubercle. But on these subjects I have not time to dwell, although their pathology, especially as illustrated by the tuberculous diseases of serous membranes, is full of interest.

* Museum of the College, No. 1512.

† Inserted in the Pathological Catalogue, vol. iii. p. 183: to it are added an account of the previous notices of the facts referred to by Dr. Jenner, Dr. Baillie, and M. Odier.

LECTURE V.

Effects of inflammation on the part in which it is seated; their generally destructive character—Softening of inflamed parts, as of brain, bones, ligaments, &c.—Fatty degeneration of inflamed parts, as in the liver and heart.—Interstitial absorption of inflamed parts, as seen in bones, cartilages, glands, &c., and in the spontaneous opening of abscesses.—Ulceration; ejection of tissues disintegrated after degeneration; supposed effects of corrosion and solution of tissues by ichorous discharges.—Gangrene.

The account of the results of inflammation in the part in which it has its seat will include the chief among its second class of effects—the destructive effects. For I believe that nearly all the effects of inflammation are injurious, if not destructive, to the proper tissues of the part in which it is seated. All the changes I shall have to describe are characteristic of defective or suspended nutrition in the parts: they present varieties of degeneration, modified by the circumstances in which they have occurred, and especially by this,—that while the changes of the proper tissues of an inflamed part are making progress with all the characters of degeneration, the lymph-products of the inflammation may be in process of either development or degeneration in their interstices, or on their free surfaces.

One of the most common effects of inflammation in an organ is a more or less speedy *softening* of its substance: and this is due not only to infiltration of it with fluid, but to a proper loss of consistency, a change approaching to disintegration, of which, indeed, it is often the first stage. Of such mere softening, some of the best examples are in the true inflammatory softening of the brain and spinal cord, in which the softened part is usually found to consist of spoiled nervous substance, together with more or less abundant granular products of inflammation. Such softening also may be found in the lungs: the peculiar brittleness and rottenness of texture, which exists with the other characters of hepatization, are evidently due to changes in the proper tissue, more than to incorporation of the products of inflammation. In staphyloma of the cornea, similar softening ensues in connection with the opacity and other changes of appearance. But, perhaps, the most striking instance of softening in inflammation (and it is the more so because the softening probably precedes the other evident signs of inflammation*) is to be found in bones. One may generally notice that an acutely inflamed bone is soft, so that a knife will easily penetrate it. Thus it may be found in the phalanges of the fingers when they partake in deep-seated inflammation, and thus, sometimes, in the neighbourhood of diseased joints. The change depends partly on an absorption of the earthy matter of the bone, this constituent being removed more quickly, and in greater proportion, than the animal matter; but the entire material of the bone is softened.

The softening of bones may permit of peculiar subsequent changes, especially of their swelling and expansion. Thus, in a remarkable case communicated by Mr. Arnott to Mr. Stanley, after excision of the corresponding ends of the radius and ulna, inflammation ensued in the shaft of the humerus, and after four months the patient died. The end of the inflamed humerus was full-red, and swollen, with an expansion or separation of the layers of its walls. And the case showed well the coincidence of absorption and of enlargement by expansion; for though the diseased humerus was thus enlarged, and contained more blood than the healthy one, yet it "was found not to weigh so much by half."†

Similar expansions of bone, with all the characters of inflammation, and such as could not have happened without previous softening of the tissues, form part of the many swollen and enlarged bones which are common in all our museums.‡ Doubtless, in many of these cases, the disease has been of very slow progress, and the separation of the several layers of the compact bone, which the specimens display, must be ascribed to their gradually altered form, as they have grown about the enlarging blood-vessels and interstitial inflammatory deposits. But in other cases the expansion has in all probability been more rapid, the softened bone yielding and extending, as

* See Kuss, as quoted by Virchow, in his Archiv.

† Mr. Stanley's Illustrations, pl. i. fig. 4, 5, 6.

‡ In the College Museum, No. 593 to 600, and 3062 to 3094; and in the museum of St. Bartholomew's, Series I. Nos. 56, 94, 113, 114, 115, 196, 197, 198, &c.

the naturally softer tissues do, in an inflammatory swelling.

The characters of a bone thus expanded are easily discerned. Its substance may be irregularly cancellous or porous; but the most striking change is a more or less extensive and wide separation of the concentric laminæ of the walls of the bone, so that the longitudinal section of the enlarged wall appears composed of two or more layers of compact tissue, with a widely cancellous tissue between them: and these layers may sometimes be traced into continuity with those forming the healthy portion of the wall. Usually, the separated layers are carried outwards, and the bone appears outwardly enlarged; but sometimes the inner layers of the wall are pressed inwards, and encroach upon the medullary tissue. In the first periods of the disease, the cancellous tissue between the separated layers of the wall has wide spaces, which are usually filled with a bloody-coloured medulla: but this tissue, like the often coincident external formations of new bone, appears to have a tendency to become solid and hard; and its fibrils and laminæ may thicken till they coalesce into a compact ivory-like substance, harder than the healthy bone.*

Again, for examples of softening in inflammation, I may adduce the softening of ligaments, such as permits that great yielding of them which we see almost always in cases of severely inflamed joints. This is not from mere defective nutrition; for it does not happen in the same form, or time, or measure, in cases of paralysis or paraplegia engendering extreme emaciation. Neither is it from the soaking of the ligaments with the fluid products of the inflammation; for it does not happen in the abundant effusions of the slighter inflammations of the joints; and when ligaments are long macerated in water they yet retain nearly all their inextensibility. It appears to be a peculiar softening, or diminished cohesion, of the proper tissue of the ligaments, the result of a defective nutrition combined with infiltration of inflammatory products.

We may see such changes in the ligaments of all joints; in the hip, in the cases of spontaneous dislocation occasionally seen, independent of suppuration or ulceration of the parts belonging to the joint; in the wrist, when the ulna after disease becomes so prominent; in the vertebræ, especially in the ligaments of the atlas and axis. But we see the effects of this softening best in diseased knee-joints and elbow-joints; and in all these cases we may often observe an interesting later change when the inflammation passes by. The ligaments softened during the inflammation yield to the weight of the the limb, or perhaps to some muscular force, and the joint is distorted. Then the inflammation subsides, the normal method of nutrition in the joints is restored, the elongated ligaments recover their toughness, or are even indurated by the organization and contraction of the inflammatory products deposited in them; but they do not recover their due position; and thus the joint is stiffened in the distortion to which its ligaments had yielded in the former period of inflammation. In the crowds of stiff, distorted, and yet not immoveably fixed joints that one sees as the consequences of inflammation, these changes must generally have happened to the ligaments:—first softening and yielding—then recovering, with induration, and perhaps some contraction, due to their atrophy and the organization of the inflammatory deposit. The cases are aggravated by similar changes in the adjacent parts; for the stiffness of such joints is not due to the ligaments alone; all the subcutaneous tissues are apt to be adherent and indurated.

The softening of the tissues of an inflamed part may be regarded as one of the instances of degeneration in the inflammatory process; and its diversity from ordinary degenerations may be ascribed to the simultaneous infiltration of the inflammatory product, and perhaps to some other circumstance we cannot at present trace or guess. But a more general and unmixed form of degeneration may, I think, be occasionally observed in the tissues of inflamed parts—namely, fatty degeneration; and this in such a manner, as to make it probable that the degeneration takes place even during the inflammation. Thus fatty degeneration of the hepatic cells appears an usual coincident of the form of inflammation which produces the so-called "brawny" liver. I think, too, that I have seen fatty degeneration of the muscular fibres in inflammation of the heart; especially in a recent case, in which the heart was punctured with a needle, and the patient died four days afterwards. The portion of the heart near the needle was more degenerate than the rest of its substance. So, also, in some instances of acute ulceration of cartilage, I have found that a fatty degeneration of the contents of the cells, together with similar degeneration or disappearance of the nuclei, constantly precedes the removal of the whole substance of the cartilage.* I am inclined,

* Abridged from the Pathological Catalogue of the College, vol. ii. p. 97.

* Similar observations are made by Dr. Redfern, especially in the third case of his excellent

therefore, to believe that this method of degeneration may occur not unfrequently in inflammation of certain parts, and may be, like the softening last described, a principal constituent of the changes preceding their complete absorption.

Both the foregoing degenerative changes in inflamed parts are favourable to complete removal by *absorption*, in which we find another example of the destructive effects of inflammation. And this absorption, which in many inflammatory conditions is a peculiarly rapid event, may affect at once the proper elements of a part, its bloodvessels, and the inflammatory products that may have been previously deposited among them.

I shall refer here only to that which has been called *interstitial absorption*; to the removal of parts from within the very substance of the tissues, as distinguished from the removal by the ejection of particles from the surface, of which I shall next speak as occurring in *ulceration*.

We may believe that such degenerations as I have just described usually precede this interstitial absorption of parts; but we cannot be quite sure of it, because we cannot see the parts immediately before absorption; or, rather, because we cannot be sure that what we see was to have been very soon absorbed. But, as I stated in the last lecture, we are justified in holding that no absorption of the living tissues can take place without previous change or degeneration of that which is to be absorbed. And we may the more certainly believe that, in the interstitial absorption which takes place in an inflamed part, degeneration and disintegration of its tissues always ensue before they are, as the expression is, "taken up," from the observation that the degenerative changes, which I have just described, are sometimes evidently the precedents of absorption. Of such interstitial absorptions of inflamed parts, we find well-marked instances in that form of ulceration of articular cartilages in which the deeper portion of the cartilage is removed, together with the adjacent osseous layer covering the head of the bone; for here we can hardly suppose that the disintegrated particles are cast out, whatever they may be in the superficial ulceration of the same tissue. Such interstitial absorption is seen, too, very well in inflamed bones. The head of a bone may be scarcely enlarged, while its interior is hollowed out by an abscess; what remains of the bone may be indurated, as by slight and tardy inflammation, but so much of the bone as was where now the abscess is, must have been inflamed and absorbed. Here, too, the evidence of absorption is completed by the similar excavations formed in bones within which cysts and tumors grow; for in these cases no other removal than by absorption seems possible.

To similar absorption of inflamed tissue we may refer the wasting and deformity that we notice in the heads of bones that have been the seat of chronic rheumatism. The best examples of this are in the head and neck of the femur; and the retention of the compact layer of bone, covering-in the wasted cancellous tissue of the shortened neck and flattened head, is characteristic of interstitial absorption, as distinguished from ulceration, by which the cancellous tissue is commonly exposed. In these cases of chronic inflammation of the bones, we may notice, also, an appearance of degeneration that precedes a peculiar mode of absorption. While the articular cartilages are passing through the stages of "fibrous degeneration," and are being gradually removed, the subjacent bone is assuming the peculiar hardness which has been termed "eburnation," or "porcellaneous" change. Now this change is effected by the formation of very imperfect bone,—of bone that has no well-formed corpuscles; and resembles the result of mere calcareous degeneration rather than of a genuine ossifying induration. And its character as a degeneration is further declared in this; that it is prone to the destructive perforating ulceration, which often gives a peculiar worm-eaten appearance to the bones thus diseased.*

With these changes in rheumatic bones we may also cite, as instances of absorption during inflammation, the changes which Mr. Gulliver† first described as apt to ensue after injuries about the trochanter of the femur. In such cases, without any appearance of ulcerative destruction, the head and neck of the femur may waste by absorption, the neck becoming shortened, and the head assuming a peculiar conical form. We might regard these effects as a simple atrophy, if it were not that they are like the effects of the more manifest inflammation in the rheumatic cases, and

essay on "Anormal Nutrition in Articular Cartilages." Perhaps, also, we ought to classify with the degenerations that may ensue in the proper tissue of an inflamed part, the ossification of the laryngeal cartilages when involved in inflammation, and the fibrous structure acquired in slightly and long inflamed muscles.

* A change, which appears to correspond with the eburnation of bone, is described by Mr. Tomes, as occurring in the part of a tooth which lies just beneath a carious cavity. In both cases the induration might suggest that it is calculated to retard the progress of the disease, but we have no evidence that it does this in an effective manner; and in the case of the bones there is every appearance that the destruction is most rapid where there is most induration.

† Edinburgh Med. and Surg. Journal, vol. lxvi. —The change is illustrated in No. 3312 in the College Museum.

that the existence of inflammation during life is often declared by the symptoms following the injury.

Again, other examples of the absorption of inflamed parts, or of parts that have been inflamed, are presented in the wasting of glands after inflammation; as in cirrhosis of the liver, in some forms of granular degenerations of the kidney, in the indurated and contracted lung after pneumonia.

No doubt, in these cases, the reduction of the organ depends, in a measure, on the contraction of the diffused inflammatory product, as it organizes; but in many cases the quantity of new tissue is extremely small (it is so in the shrivelled granular kidney); and, in all the cases, we may well doubt whether the contraction of organizing lymph would produce such extensive and uniform absorption of the proper substance of an organ, if there were not a previous condition favouring the absorption. The most probable explanation of these cases seems to be, that as, in the early periods of the inflammation, the softening and the degeneration of the inflamed tissues coincide with the production of the lymph; so, as the inflammation subsides, and subsequently, the absorption of the degenerated tissues may often coincide with the full organization and contraction of the lymph. And it is altogether most probable that these events are independent, though concurrent; that each occurs as of itself, not as the cause or consequence of the others.

To all these cases must be added the fact of the absorption of the blood-vessels, and other accessory apparatus, of the inflamed tissues. The absorption of the absorbents themselves must coincide with that of the tissues. What a problem is here! These, that had once been the apparatus maintaining life, that had been adjusted to its energy and fashion, now, as it fails, remove themselves in adaptation to its failure. How can this be? We can only guess that its method is just the reverse of the method of formation; that, as in growth the blood-vessels and lymphatics follow in the course of evolution of the growing parts, opening and extending into each new part as it forms, so, in decrease, they follow, and closing-in harmoniously with the general involution, mingle their degenerate materials with those of the tissue, and are absorbed by the nearest remaining streams of blood.

Once more; not only the original elements of the tisues may be absorbed, but, even more rapidly, the new-formed products of inflammation. We have the best example of this, as well as, indeed, of many of the facts which I have been mentioning, in the spontaneous opening of a common abscess; which, though it be so common a thing, I will venture to describe here.

Let us suppose the case of an abscess formed in the subcutaneous tissue; of such an one as may have had its origin in lymph infiltrated through a certain area of the tissues, and forming therein a hard circumscribed inflamed mass. Of this lymph we may suppose the greater part degenerating into pus. It may begin to do so at a central point,—the point at which the conditions of nutrition are the most impaired: or the suppuration may begin at many points at once, and, thence extending, the several collections of matter may be fused together. Sometimes masses of the infiltrated and softened tissue are thus detached and cast loose in the cavity of the abscess (as in these specimens.)* But at length we may suppose all the central portion suppurated, while the peripheral part, as happens usually in chronic abscesses, may be more organized, may acquire blood-vessels, and may thus assume the character of a granulation-layer, and form the proper wall of the abscess.

The pus of such an abscess as this will contain, probably, besides its proper constituents, some of the disintegrated tissue of the part in which it has its seat. We cannot, indeed, be quite sure of this; for it may be, that while the lymph is being formed, or being converted into pus, the proper tissue of the part may be undergoing absorption; and although, in the pus of abscesses thus formed, we often find abundant molecular and granular matter, yet this may be the débris, not of the tissue, but of the lymph-cells or pus-cells, or of the fibrine which may have coagulated with the lymph-cells. We cannot, I think, be sure on this matter; but we may be sure that the circumscribed portion of tissue, in which such an abscess as I am describing has its seat, degenerates, and is either absorbed, or else disintegrated so as to mingle more or less of its substance with the pus.

In such an abscess, moreover, we often find a layer of cells, spread out like a thin yellowish-white membrane, on the interior of the wall. They are only like lymph-cells or pus-cells, not yet mingled with the rest of the contents of the abscess: but they have been made to seem more important by being called a 'pyogenic membrane,' and by its being sometimes implied that it is their work to secrete the pus. But the existence of such cells is far from constant in abscesses, and we cannot suppose a special membrane necessary for the formation of pus, while we see the best examples of it formed on

* College Museum, 113, 114.

granulating wounds, and on mucous surfaces, which have no such 'pyogenic' membrane.

The abscess thus formed has a natural tendency to open, unless all the inflammation in which it had its origin subsides. Inflammation appears to be essential to the spontaneous opening of abscesses; for, where it is absent, the matter of chronic abscesses will remain, like the contents of any cyst, quiet for weeks, or months, or years; and when in chronic abscesses, or in cysts, inflammation ensues through the whole thickness of their coverings, it is usually certain that their opening is near at hand. This difference between acute and chronic abscesses makes it very doubtful whether the inflammation of the coverings of an abscess can be ascribed to any local influence of the pus. But to whatever it may be ascribed, we may refer to this inflammation the comparatively quick absorption of the integuments over the collection of matter: and thus the fact, however we may account for it, that the integuments are more prone to inflammation, and more actively engaged in it, than the other tissues about an abscess are, may be used to explain the progress of matter towards the surface. Possibly—though this I think is much less probable—the tissues between an abscess and the surface may, after the degeneration which accompanies their degeneration, be disintegrated, and may mingle their molecules with the purulent contents of the abscess. But in favour of the belief that they are absorbed we have the evidence of analogy; for just the same thinning and removal of integuments takes place when they inflame over a chronic abscess, with a thick impenetrable cyst, or over an encysted or even a solid tumor.* Here absorption alone is possible; and the cases are so similar to the ordinary progress of abscesses, that I think we may assign all the changes of the integuments over these to the same interstitial absorption.

As the absorption proceeds, the integuments grow not only thinner, but softer, and more yielding. And this softening is worth notice, because one might suppose that as pus accumulates, so the integuments over it would become tenser and more resisting. It is, probably, in great measure, such a softening as I have already spoken of in degenerating inflamed parts; but it may be also due, in some degree, to such a change as that to which Mr. Hunter refers as "the relaxing or elongating process." He used to show this specimen†, and say of it in his lectures, "This preparation represents the front of a chest which contained an aneurism of the aorta; and here, on the right side, you see an instance of the elongating process, the cartilages being bent outwards, or elongated, to adapt themselves to its figure." Of the same process he says elsewhere,* "Besides these two modes of removing whole parts, singly or together [that is, besides the interstitial and the progressive absorption], there is an operation totally distinct from either; and this is a relaxing and elongating process carried on between the abscess and the skin, and at those parts only where the matter begins to point. It is possible that this relaxing, elongating, and weakening process, may arise in some degree from the absorption of the interior parts; but there is certainly something more, for the skin that covers an abscess is always looser than a part that gives way from mere mechanical distension, excepting the increase of the abscess is very rapid.

"That parts relax or elongate without mechanical force, but from particular stimuli, is evident in the female parts of generation, before the birth of the fœtus; they become relaxed prior to any pressure. The old women in the country can tell when a hen is going to lay from the parts becoming loose about the anus."

I have quoted the whole passage, because I believe that more recent researches have done nothing towards either improving the description or explaining the fact, except in so far as they make it probable that the change is due in a measure to the inflammatory softening of the pointing skin.

While these changes of degeneration and removal, of softening and relaxing, are ensuing in the cutis over such an abscess as I have described, we commonly notice that the cuticle separates, leaving the very point, or most prominent part, of the abscess bare. The cuticle is not raised as in a blister, but peels-off like dead cuticle; and we may believe that it is dead, partaking in the failure of nutrition in which all the parts over the abscess are involved, and being removed as a dead, not as a merely degenerated, part: for thus cuticle is always removed.

At length, after extreme thinning of the integuments, they perish in the centre of the most prominent part.—Sometimes the perished part becomes dry and parchment-like, with a kind of dry gangrene; but much more commonly a very small ordinary slough is formed, and the detachment of this gives issue to the purulent matter. The discharge is usually followed by a

* As in No. 121, College Museum.

† College Museum, No. 122; and Catalogue, vol. i., p. 53.

* On the Blood, &c. Works, vol. iii. p. 477.

more or less complete cessation of the inflammation in the integuments, and then the wall of the abscess, having the character of a cavity lined with healthy granulations, heals.

Such appears to be the ordinary course of an abscess; and I venture to hope that the numerous principles of inflammation that it illustrates will justify my having used so much time in describing it.

I proceed now to the consideration of *Ulceration*, as one of the effects produced by inflammation in the proper tissue of the inflamed part.

I need hardly say that, ever since Hunter's time, confusion has existed in the use of the terms employed for various kinds or methods of absorption and ulceration. Of all that Hunter wrote, nothing, I think, is so intricate, so difficult to understand, as his chapter on ulcerative inflammation; and much of the obscurity in which he left the subject remains. For a general consideration of the subject, it may suffice to speak (as I have done) of the removals of the particles of inflamed parts, which are not on an open or exposed surface, as the "interstitial absorptions" of inflamed parts. Then, the term "ulceration" may be employed to express the removals of the superficial or exposed particles of inflamed parts. If these superficial particles may be supposed to be absorbed, the process of removing them may be termed "ulcerative absorption;" but if it is more probable that their removal is effected entirely by ejecting them from the surface of the inflamed part, then the term "ulceration" may sufficiently express this ejection, and will stand in stronger contrast to the "interstitial absorption" of the particles that are not so ejected.

I have lately referred to the uncertainty whether, as the cavity of an abscess enlarges, the tissues that are removed from the inner surface of its boundary-walls are absorbed, or are disintegrated and mingled with its fluid contents; in other words, whether they are absorbed or ejected. The same uncertainty exists in the case of ulceration. Is the enlargement of an ulcer effected by absorption of its boundaries, or by the gradual detachment and casting-off of particles from their free surface? Both methods of enlargement may, perhaps, in some cases, ensue; but the probabilities are in favour of the enlargement being, as a rule, effected by the ejection of particles.

Thus:—1. Parts to be removed from a surface are generally cast-off rather than absorbed, as cuticles of all kinds are, and the materials of secretions; so that, by analogy, we might assume that the particles of the surface of a spreading ulcer would also be cast-off.

2. The materials of the ulcerating tissue may be sometimes found in the discharge from the ulcer. In most cases, indeed, this is impossible; but perhaps it is so only because, when the tissues are degenerate, and broken-up, or decomposed and dissolved, we have no tests by which to recognise them. In the case of bone, however, some of the constituents of which are not so easily disguised, the ejected materials may be found. In one of his lectures delivered in this theatre, Mr. Bransby Cooper mentioned that, while in pus from soft parts only traces of phosphate of lime were found, the pus from around diseased bone contained in solution nearly $2\frac{1}{2}$ per cent.* A similar, but less complete observation, had been made by Mr. Thomas Taylor,† and by v. Bibra;‡ and we may be nearly sure that the phosphate of lime was, in these cases, some of what had existed in the diseased bone.

3. It strengthens this belief to observe, that, in many cases, small fragments of bone and other tissues are detached and cast-out with the fluid secreted from the ulcerating part. These, indeed, when they are not fragments of tissue detached by ulceration extending around them, are good examples of the transition that may be traced from ulceration to sloughing or gangrene of parts, between which, if ulceration be always accomplished by ejection, the only essential difference will be one of degree: the ulceration being a death and casting-off of invisible particles of a tissue, while gangrene implies the death and casting-off of visible portions.

4. And it may be proved of many that we call ulcers that they begin as sloughs which are cast off, and leave the ulcerated surface beneath. We may often see this, on a large scale, in the instances of what

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* Medical Gazette, May, 1845.

† Stanley, on Diseases of the Bones, p. 89.

‡ Chemische Untersuchungen verschiedener Eiterarten, p. 85. It may seem that, in these cases, a further proof is needed that the quantity of bone-earths discharged with the pus is proportionate or equal to the quantity lost by the ulcerating bone. But this proof may be neither possible nor necessary; for if what has been already said, of the conformity of the properties of inflammatory and reparatory products with those of the tissues from which they are produced, be true, then will also pus from diseased bone possess more bone-earths than pus from any other tissue, even though the bone be not ulcerating. Granulations upon bone doubtless contain more bone-earths than those from soft parts, and they may ossify: now the relation of pus to granulations is commonly that of degenerating cells to the like cells developing; therefore we might expect that pus from bone, like granulations from bone, will contain a large proportion of bone-earths, independent of what may be derived from the ulceration of the bone.

are called sloughing ulcers; but Dr. Baly has proved it for a much wider range of cases, in his observations on dysentery, in which he has traced, how even the smallest and the most superficial ulcers of the intestine are preceded by the death and detachment of portions of the mucous membrane, with its covering of basement-membrane and epithelium.*

From these considerations, we may hold it as probable that ulceration is, usually, the result of the detachment of dead portions or molecules of a tissue, and that the substance removed in the process is not absorbed but ejected. There are, indeed, some cases which may make us unwilling to admit, at present, that all ulceration is by ejection; such as those of bone ulcerating under cartilage, or in the rapid extension of inflammation within it, or such as the spreading ulceration of the vertebræ, or the heads of bones, that is not attended with external discharge of fluid. These may interfere with the universality of the rule, but not with its generality.

But, if we may believe that the removal of a tissue by ulceration is generally effected by ejection of its substance, the question may be asked, in what form is it ejected? Dr. Baly's observations enable us to say that, in the first instance, a visible slough is detached, a portion of the tissue dying and being disconnected from the adjacent living tissue. But, after this is done, when an ulcer enlarges, or extends and spreads, is the material of the tissue still removed in visible sloughs or fragments? Certainly it is so sometimes; for we may find little fragments of bone in the discharge from ulcerating bone, especially in strumous ulceration. But in other cases we have no evidence of this kind; we cannot detect even microscopic fragments of tissues in the discharges, and we must suppose that they are removed, in a state of solution or of molecular subdivision, in the discharge from the diseased part.

To speak of the solution of tissues in the discharges of ulcers may seem like the revival of an old error long since disproved. But though the expression may be revived, it is with a new meaning. The proof has, truly, been long completed, that healthy tissues, even though they be dead, cannot be dissolved in pus, or any such discharge; but the tissues that bound or form the walls of a spreading ulcer are not healthy; they are inflamed, or otherwise diseased and degenerate; and they may now be soluble in fluids that could not dissolve them while they were sound. Insolubility is as great an obstacle to absorption as to ejection in discharges; no tissue can be absorbed without being first so far changed as to be soluble in fluids with which it was before in contact and unharmed. Therefore, whether we hold the ordinary spreading of an ulcer to be by absorption of its boundaries, or ascribe it to their ejection, we must, in either case, admit that they are first made soluble. And if this be admitted, then it is most consistent with analogy, and most probable, that the extension of an ulcer, independently of sloughing, is accomplished by the gradual degeneration of the tissues that form its walls, and by their being either disintegrated and cast-off in minute molecular matter, or else dissolved and ejected in solution in the discharges from the ulcer.

The solution here spoken of is such as may be effected by the fluid discharged from any spreading ulcers; and we may doubt whether all discharges from ulcers possess a *corroding* property, such as Rokitansky seems to ascribe to them, and such as he considers to be the chief cause of the extension of all ulcers. We may doubt, I say, whether all ulceration can be described as a corrosion or erosion of the tissues by ichor; but, on the other side, we cannot well doubt that the properties of the discharge from an ulcer, or a sloughing sore, may have a great influence in accelerating the degeneration and decomposition, and thereby the solution, of the tissues that form its walls or boundaries. Many ichorous discharges from ulcers inflame and excoriate the parts over which they flow; and one constituent of inflammation is the defective nutrition of the proper elements of the affected tissue. Many such discharges, also, are in an active state of decomposition; and their contact with the tissues cannot but have some tendency to excite decomposition in them; a tendency which the tissues will be the less able to resist, in the same proportion as they are already feebly maintaining themselves, or as they have been moved by inflammation from their normal conditions, and their normal tenacity of composition.

On the whole, then, we may conclude, respecting the process of ulceration, that its beginning is usually the detachment of a slough, or portion of dead tissue, by the removal of the layer of living tissue that bounded it; that the spreading of an ulcer, independent of such visible sloughing, is effected by the tissues that bound it becoming degenerate, and being detached in minute particles, or molecular matter, or being decomposed and dissolved in the fluid discharge or ichor; and that this spreading may be accelerated by the influence of the discharge itself, which may inflame the healthy tissues that it rests on, and may exercise a decomposing "catalytic"

* Gulstonian Lectures. Medical Gazette, 1847.

action on those that are inflamed already.

I have already said that the products of inflammation are commonly removed, in ulceration, together with the elements of the tissues in which they are deposited. And all that has been said of the changes that the tissues undergo previous to ejection or solution, may be said, also, of the products of the inflammation which commonly precedes and accompanies the ulcerative process.

I need hardly say that we have no knowledge by which to explain the peculiar and characteristic forms of certain ulcers. We seem wholly without a guide to such knowledge; but the existence of such specific forms is conclusive against the supposition that the extension of an ulcer is entirely due to corrosion by an exuded fluid. Such a fluid would act uniformly, unless the various effects of disease on the tissues bounding the ulcer should make them variously amenable to its influence.

The last enumerated effect produced by inflammation in the tissues in which it is seated is gangrene, mortification, or sloughing.

In the usual enumeration of the effects of inflammation, it is commonly implied that the processes of effusion of lymph, suppuration, ulceration, and mortification, may be taken as the expressions of so many successive degrees of severity of the morbid process. But this is far from being unconditionally true. The exciting cause of the inflammation, the tissue affected, the condition of the blood, the general amount of vital force, and the degrees in which the bloodvessels can adapt themselves to the transit of various quantities of blood; all these, and, perhaps, several other things, have as marked an influence as the severity of the disease has in determining the result of an inflammation to be gangrene of the affected part. It may be nearer the general truth, to say that the probability of gangrene ensuing in inflammation is proportionate to the sum of the intensity of the disease *plus* the debility or defective vitality of the affected part, whether that debility have a general or a local origin. But, indeed, in any case of gangrene that may be regarded as inflammatory, it is very hard to say what may be ascribed to the inflammatory process, and how that process affects the issue, so great are the number, and so diverse the natures, of the several morbid conditions that are from the first, or in succession, involved. Certainly, it is not possible to speak clearly of inflammatory gangrene without constant reference to many other forms of local death which lie far beyond the range of these lectures. For this reason, and because a separate series of specimens in the Museum is devoted to the illustration of the death of parts, I shall here conclude the account of the phenomena and effects of inflammation.

LECTURE VI.

Nature and Causes of Inflammation. Limitation of the term 'increased action' as applied to the process of inflammation. Indications of defective nutrition, and defective exercise of the vital forces; in the effects described in the preceding lecture, and in the low organic characters of the inflammatory products, and in the conditions preceding the process. Peculiarities by which the inflammatory mode of nutrition is generally distinguished from hypertrophy, atrophy, and the production of new growths, such as tumors.

Proximate causes of inflammation traced to qualitative changes of the necessary conditions of normal nutrition; as, 1. *of the Blood-vessels:* 2. *of the Blood; especially in inoculable diseases, and specific inflammations; explanation of the localization of a general disease of the blood:* 3. *of the Nervous force, as affecting the size of the blood-vessels, and as having a share in the plasturgic forces:* 4. *of the proper tissues of the inflamed part.*

An examination of the nature of the process of inflammation may best be made in the form of a comparison of its effects with those of the normal process of nutrition. And this comparison may be drawn with two principal views; namely, to determine, 1st—how the effects of inflammation differ, in respect of *quantity*, from those of the normal process; and 2nd, how they differ from the same, in respect of *quality* or *method.*

The decision on the first of these points may seem to be given in the term 'increased action,' which is commonly used, as synonymous with inflammation. As used by Mr. Hunter, this term was meant to imply that the small vessels of an inflamed part are more than naturally active, in formation or absorption, or in both these processes. This is, probably, the meaning still generally attached to the term by some; while, as employed by those who believe the vessels are only accessories in the work of nutrition, the expression 'increased action' may be used to imply merely increased formation, or increased absorption. In either, or in any, meaning, however, the term seems to involve the idea of an increased exercise of the vital forces, *i. e.* of those forces through the operation of which the various acts of organic formation are accomplished. But, if 'increased action' is to imply this, the description of the process and effects of inflammation shews that the term cannot be properly used, without some limit or qualification.

If we consider the quantity of organic formation effected during the inflammatory process, in the proper substance of the inflamed part, it is evidently diminished. All the changes described in the last lecture are signs or results of diminished or suspended nutrition in the tissues of the inflamed part: they are all characteristic of atrophy, degeneration, or death. The tissues become soft or quite disorganized; they are relaxed and weakened; they degenerate, and remain lowered at once in structure, chemical composition, and functional power; or else, after degeneration, they are absorbed, or are disintegrated, or dissolved, and cast out; they die in particles or in the mass. During all the process of inflammation, there is no such thing as an increased formation of the natural structures of the inflamed part; they are not even maintained; their nutrition is always impaired, or quite suspended. It is only after the inflammation has ceased that there is an appearance of increased formation in some of the lowly organised tissues, as the bones and cellular tissue.

So far, then, as the proper substance of the inflamed part is concerned, there appears to be decreased action; that is, decreased formation. There may be, indeed, an increased absorption; but this is also, in one sense, characteristic of decreased exercise of vital force; since all absorption implies a previous degeneration of the part absorbed. Nor can we justly call this, in any sense, 'increased action,' till we can show how absorption is an act of vessels.

On the whole then, we may conclude, thus far, that one of the constituents of the inflammatory process, one of the characters in which it differs, in respect of quantity, from normal nutrition, is a defect in the nutrition of the proper substance of the inflamed part.*

* This has been clearly maintained by Dr. Carpenter. See Br. and For. Med. Rev. July, 1844.

But it is characteristic of inflammation, that, while the inflamed part itself suffers deterioration, there is a production of material which may be organized. Here, therefore, may be an evidence of increased formation, of increased action.

Doubtless, in relation to the productive part of the inflammatory process, the expression 'increased action' may be in some sense justly used; for the weight of an inflamed part, or of the material separated from it, may be much increased by the formation of organized matter. But the quantity of organized matter formed in an inflammation must not be unconditionally taken as the measure of increase in the exercise of the vital forces: for it is to be observed, that the material formed presents only the lowest grades of organization, and that it is not capable of development, but rather tends to degeneration, so long as the inflammation lasts.

It may be but a vague estimate that we can make of the amount of vital force exercised in any act of formation; yet we may be sure that a comparatively small amount is sufficient for the production of low organisms, such as are the fibrinous and corpuscular lymphs of inflammation. The abundant production of lowly organised structures is one of the features of the life of the lowest creatures, in both the vegetable and animal kingdoms. And, in our own cases, a corresponding abundant production is often noticed in the lowest states of vital force; witness the final inflammations, so frequent in the last stages of granular degeneration of the kidneys, of phthisis, of cancer, and other exhausting diseases. In all these, even large quantities of the lowly organised cells of inflammatory lymph may be formed, when life is at its last ebb. And with these cases correspond those that show the most rapid increase of tubercle and cancer, and other lowly organised tumors, when the health is most enfeebled, and when the blood and all the natural structures are wasting.

From these considerations, we may conclude that the productive part of the inflammatory process is not declaratory of the exercise of a large amount of vital, or organising, force; and this conclusion is confirmed by observing that development, which always requires the highest and most favoured exercise of the powers of organic life, does not occur while inflammation lasts. The general conclusions, therefore, may be, as well from the productive, as from the destructive, effects of the inflammatory process, that it is accomplished with small expenditure of vital force, and that even when large quantities of lymph are formed, such an expression as "increased action" cannot be safely used, unless we can be sure that the defect of the formative power exercised in the proper tissue of the inflamed part is more than counterbalanced by the excess of power manifested in the production and low organisation of lymph.

It may be said that the signs of inflammation are signs of increased action. But these are fallacious, if, again, by increased action be meant any increased exercise of vital force. The redness and the swelling of an inflamed part declare the presence of more blood; but this blood moves slowly; and it is a quick renewal of blood, rather than a large quantity at any time in a part, that is significant of active life. An abundance of blood, with slow movement of it, is in no case characteristic of activity in a part; it more often implies the contrary, as in the erectile tissues, and the cancellous tissue of bones.

The sign of heat in the inflamed part is equally fallacious. The source of the locally increased heat cannot, I believe, be satisfactorily explained. This phenomenon of inflammation is involved in the same difficulty as are all those that concern the local variations of temperature in the body: difficulties which the doctrines of Liebig, however good for the general production of heat, are quite unable to explain. But, from the fact that the general supply of heat in our bodies is derived from oxydation or combustion of wasted tissues or of surplus food, we may assume that, in local augmentation of heat, the source is rather from some similar destruction of organic substances, than from increased formation of them. This can, indeed, be only assumed; but, if there be little evidence for it, there is as little for any assumption that the increased heat of an inflamed part is an indication of an increased formative action. The full heat of an actively growing part may be compared with the high temperature of one which is the seat of "determination" of blood, or of "active congestion;" for, in both cases, the heat is high because the blood, brought quickly from the heart, is quickly removed; but, in an inflamed part, the blood is not so renewed; it moves more slowly.

In thus endeavouring to estimate the difference between the normal and the inflammatory modes of nutrition, in regard to the quantity of formative or other vital force exercised in them respectively, I have also stated the chief differences between them in relation to the quality or method of nutrition. The most general peculiarity of the inflammatory method, in its simplest form, is the concurrence of these two distinct and independent, though usually coincident, events: namely, 1st, the impairment or suspension of the nutrition of the proper

substance of the inflamed part: and 2nd, the effusion, from the blood, of a material more than sufficient in quality for the nutrition of the part, but less than sufficient in its capacity of development.

The predominance of one or the other of these components is the ground of some of the chief varieties in the forms of inflammation; distinguishing, especially, the adhesive and suppurative inflammations, in which the formative part predominates, from the ulcerative and gangrenous, in which the destructive part prevails. But in all cases, the two components of the process are in certain measures combined, and their combination establishes the chief differences between the inflammatory and every other mode of nutrition in a part. Thus, from all the forms of mere atrophy or degeneration, the inflammatory process is distinguished by the production of the lymph, which may be organising, even while the proper tissue of the inflamed part is in process of atrophy, degeneration, or absorption. So far as the tissues inflamed are concerned, some inflammations might be classed with atrophies or degenerations; but the concurrent production of lymph is distinctive of them.

On the other side, the inflammatory mode of nutrition is distinguished from all the forms of hypertrophy by the failure of the nutrition of the inflamed part itself. So far as mere production and formation of organisms are concerned, some inflammations might be paralleled with hypertrophies; but the organisation of the lymph falls short of that proper to the part in which it is exuded; and the substance of the part, instead of being augmented, is only replaced by one of lower organisation.

And, lastly, from the production of new growths, such as tumors, the inflammatory process is distinguished by this;—that its organised products, though like natural tissues of the body, are usually infiltrated, fused, and interwoven into the textures of the inflamed parts; and that, when once their development is achieved, they have no tendency to increase in a greater ratio than the rest of the body.

I am well aware that these can be accepted as only the generally distinguishing characters of the simplest inflammatory process. Cases might be easily adduced in which the border-lines are obscured; inflammations confounded on one side with atrophies, on another with hypertrophies, on a third with tumors. But the same difficulties are in every department of our science; yet we must acknowledge the value of general distinctions among diseases even more alike than these are.

The case that I have chosen for illustrating the general nature of the inflammatory process is one representing the disease in its simplest form and earliest stage, manifesting only the formation of lymph, and such a change as the softening or absorption of the inflamed part. This is but the beginning of the history: but, if the inflammation continues, or increases in severity, all that follows is consistent with this beginning; all displays the same double series of events, the same defective nutrition of the part, and the same production of low organisms. But these additions are observed; the part is more and more deteriorated, and perishes in the mass, or in minute fragments; the newly-organised products, not finding the necessary conditions of all nutrition, partake in the degenerative process, and, instead of being developed, are degenerated into pus, or some yet lower forms, or perish with the tissues in which they are imbedded.

Respecting, now, the causes of inflammation, I shall not say more of its exciting causes than that, from the external ones, which alone we can at all appreciate, we may derive a confirmation of the opinion I have expressed concerning the nature of the process. They are such as would be apt to produce depression of the vital forces in a part; all being, I think, such as, when applied with more severity, or for a longer time, will lead, not to inflammation, but to the death of the part. If a certain excess of heat will inflame, a certain yet greater heat will kill: if some violence will inflame, a greater violence will kill: if a diluted chemical agent will only irritate, the same concentrated will destroy the part. The same may be said, I think, of cold, mechanical injury, and all the other external exciting causes of inflammation. I am aware that other explanations of their action are given; but none seems to me so simple, or so consistent with the nature of the process that follows them, as this, which assumes that they all tend (as it may be said) to depress the vital forces exercised in the affected part: they may be stimulants or excitants of the sensitive nerves of the part, but they lead to the opposite of activity in its nutritive processes.

The proximate causes of inflammation appear to be various perversions of the necessary conditions of healthy nutrition in a part; that is, morbid changes in either the supply of blood, the composition of the blood, the influence of the nervous force, or the condition of the proper substance of the inflamed part. Any one or more of these four conditions of nutrition being changed in quality, the result appears to be an inflammation. A change in quantity more usually produces either an excess or deficiency of nutrition in the part, or

some process different from inflammation. Thus, a diminution or withdrawal of the blood, without alteration of its quality, is usually followed by atrophy, degeneration, or death: a mere increase of blood in a part may produce hypertrophy, or something more nearly resembling inflammation, yet falling short of it. Similar effects may ensue from a mere increase or decrease, or abstraction, of nervous force. Change in the quality, whether with or without one in the quantity, of the conditions of nutrition, appears essential to the production of the phenomena of inflammation.

I will endeavour now to show that inflammation may follow such perversion or qualitative change in each of the conditions of nutrition, even though all the rest of them remain in their normal state: selecting, for this purpose, such cases of inflammation as we may trace proceeding, in the first instance, from the uncomplicated error of a single condition of nutrition.

1st. Inflammation may perhaps be produced—it certainly may be in some measure imitated—by changes in the blood-vessels; changes attended with alteration of their size, or their permeability, or the other qualities by which they affect the supply of blood to a part. This may be concluded from the similarity to some of the phenomena of inflammation which may be observed in certain cases of mechanical obstruction to the venous circulation. In a case of ascites from diseased heart or liver, the peritoneum often contains coagula of fibrine floating free in the serum, though no organ may present appearances of having been inflamed. In such a case, moreover, I have found the fibrine developing itself in the form of nucleated blastema, even while floating free. In another case of mechanical dropsy, I have found the fluid of anasarca in the scrotum containing abundant lymph-corpuscles, like those in the fluid of an inflammatory effusion. Such as these are the cases through which mechanical congestions of blood connect themselves with inflammation. And if to these we add the constancy of increased vascularity among the phenomena of inflammation, they may be sufficient to make us believe, that disturbances in the circulation of a part may produce some of the principal phenomena of inflammation, even though all the other conditions of nutrition are, in the first instance, unchanged. But I know no other good evidence for the belief; and I think we should not lay much stress on these cases, since they display an imitation of only one part of the process of inflammation, namely, the production of organisable matter. The nutrition of a part with obstructed circulation suffers but a trivial disturbance, in comparison with that which would accompany an inflammation with an equal amount of hindrance to the movement of the blood. I should therefore be cautious of regarding these effusions in mechanical obstructions of blood as more than partial imitations of the inflammatory process. So far as the effusion in an inflammation depends on the altered mechanical relations of the blood and vessels of a part, so far may similar alterations produce effects imitating those of inflammation; but I much doubt whether any change whatever in the circulation of a part, however produced, can alone produce or alone maintain the phenomena or effects of inflammation. I believe that the disturbances of the circulation are no more adequate to the explanation of inflammation, than the normal movements of the blood are adequate to the explanation of the ordinary process of nutrition.

2. We may speak much less equivocally of the influence of the state of the blood itself in determining inflammations; for there can be little doubt that a very great majority of the so-called spontaneous or constitutional, as distinguished from traumatic, inflammations, have herein their origin. We might anticipate this from the consideration that, in normal nutrition, the principal factors are the tissues and the blood in their mutual relations: but we have better evidence than this, in cases of local inflammations occurring in consequence of general diseases of the blood. Some instances of this are clearly proved, as, *e. g.*, in the cases of eruptive fevers, when the presence of morbid materials in the blood is proved by the effects of their transference in inoculation. Scarcely less thoroughly demonstrated are the cases of rheumatism and gout, of lepra, psoriasis, herpes, eczema, erysipelas, and other such affections, whose constitutional nature—in other words, whose primary seat in the blood—all readily acknowledge in practice, if not in theory. Now, in all these cases, local inflammations are the external signs of the general affection of the blood: and I apprehend that if any difficulty be felt in receiving these as evidences that the morbid condition of the blood is the cause of the local inflammation, it will be through doubt whether a general disease of the blood—a disease affecting the blood sent to every part—can produce peculiar phenomena of disease in only certain small parts or organs. But this local effect of a general disease of blood has its illustration in some of the sure principles of physiology; especially in this—that the presence of certain materials in the blood may determine the formation of appropriate organisms, in

which they may be incorporated.* Thus, when one kidney is removed or destroyed, the other will acquire a greater size, sufficient for the discharge of the necessary quantity of urine. Now we know that the principal materials of urine exist ready-formed in the blood; that, being taken from the blood, they are incorporated in the secreting cells of the kidney, and are for a time enclosed in them, and enter into their composition; and that, finally, they are discharged into the excreting tubes from these cells, not by mere filtration. We may, therefore, safely hold that, when one kidney is lost or spoiled, more renal cells are formed in the other, *because* more of the constituents of urine are in the blood: in other words, that the presence of these constituents in the blood that is carried to every part determines the formation of appropriate organs in one part of the body; in which organs these constituents may be incorporated. Nor is this the only case. Certain medicines, especially diuretics, are separated from the blood by only certain organs; they must, for this separation, be embodied in those organs, and while embodied they may excite inflammation; as cantharides, turpentine, and the like, do. Abundant hydro-carbon principles in the blood will bring about the formation of abundant fat cells, in which they may be enclosed. The accumulation of sap in the branch of a tree that has been *ringed* will determine the organisation of abundant fruit; and many similar cases might be cited.

It is in exact parallel with these facts in physiology, that in certain general diseases of the blood, organs are formed, as the products of inflammation, within which the specific morbid material is incorporated. Thus, in small-pox, cow-pox, primary syphilis, and whatever other diseases may be transferred by inoculation, the morbid material from the blood is incorporated in the fluid and corpuscular products of inflammation, which are enclosed within the characteristic vesicle or pustule; just as, in the cases already cited, the constituents of urine or of medicines are incorporated in the renal cells, which are formed within the substance of the kidney; or just as the constituents of sap are incorporated in fruit.

In the cases of disease produced by a demonstrable virus, we have all the evidence that can be necessary to prove the principle I am contending for—namely, that a general disease of the blood may determine a local inflammation in one or more circumscribed portions of a tissue. And the analogy is so close, that I think we need not hesitate to receive the same explanation of other inflammations, which I have cited as occurring during morbid conditions of the blood. For although we cannot, by inoculation, prove that a specific morbid material of such a disease as herpes, or rheumatism, has been incorporated in the inflammatory products, yet we find great probability hereof in the many analogies which these diseases present to the inoculable diseases, in their whole history, and, especially, in the decrease of general illness which ensues on the full manifestation of the local inflammation.

If it be asked why a morbid material is determined to one part or tissue rather than another, or why, for example, the skin is the normal seat of inflammation in small-pox, the joints in rheumatism, and so on, I believe we must say that we are, on this point, in the same ignorance as we are concerning the reason why the materials of sweat are discharged at the skin, those of urine at the kidneys, of bile at the liver, or why the greater part of the fibrine is incorporated in the muscles, and of gelatine in the bones. We cannot tell why these things are so; yet we believe them, and our belief has practical advantages. So may the belief that a great majority of the so-called spontaneous local inflammations are the consequences and manifestations of certain morbid conditions of the whole mass of blood; and that when such conditions depend on the presence of any specific material in the blood, a portion, or the whole, of that material is usually incorporated in the products of the inflammation, and in them is separated from the blood.

But, again, it may be said, that if this be granted, still we need some explanation of the fact that the morbid condition of the blood does not influence the whole extent of any given tissue, but only portions of it. In the secretion of urine, it may be believed that the whole kidney is affected and works alike; but in the assumed separation of the virus of small-pox, only patches of the skin are the seats of pustules; in vaccinia and primary syphilis, only a single point; in secondary and tertiary syphilis, a certain, but often disorderly, succession of various parts, and so on.

It must be admitted that many of the facts here referred to cannot yet be ex-

* This principle was fully discussed in the Lectures on Nutrition delivered in 1847, and published in the MEDICAL GAZETTE of that year. And it appears to be capable of even much wider extension than was then assumed. For example, it supplies the best theoretic expression of the origin of cancerous, and similar tumors. The cells or the tissues of these growths, we may believe, are formed so as to comprise or enclose specific morbid materials generated or inserted in the blood. That each such morbid material should have, for its appropriate habitation, cells and a mass of peculiar form and appearance, is only consistent with normal rules of formation.

plained; but such difficulty of explanation affords no warrant for a denial of the theory, especially since we are able, consistently with this theory, to point out some of the conditions that determine the locality in which a general disease of the blood will manifest itself by inflammation. In some instances, it is evident that the localization of a general disease of the bood is determined by a previous condition, such as we may call a weakened or depressed condition—a state of already impaired nutrition—in some one part. For instance, suppose a stream of cold air is impelled on some part, say the shoulder, of a person disposed to rheumatism, it determines, as a more general exposure to cold might do in the same person, the rheumatic state of the blood, with all its general symptoms; but it determines, besides, the part in which that rheumatic state shall manifest itself first or alone. The depressed nutrition of the chilled shoulder makes it more liable than any other part to be the seat of inflammation excited by the diseased blood.

Or, again, when a virus is inserted, as in all cases of poisoned wounds, the local inflammation produced by the disease with which the whole blood is infected will commonly have its seat in the wounded part. The virus must have produced some change in the place in which it was inserted, as well as in the whole mass of the blood. These cases are, probably, only examples of the general rule, that a part whose natural force of nutrition is in any way depressed, will, more than a healthy part, be liable to become the seat of chief manifestation of a general blood-disease. Thus, a part that has been the seat of former disease or injury, and that has never recovered its vigour of nutrition, is always more liable than another to be the seat of local manifestation of blood-disease: it is, as they say, *a weak part*. Thus the old gouty or rheumatic joint is apt to receive the brunt of the new attack. And the same may happen in a more general way. A man was under my care with chronic inflammation of the synovial membrane of his knee, and general swelling about it: he was attacked with measles, and the eruption over the diseased knee was a diffused bright scarlet rash. A patient under Dr. Budd's care had small-pox soon after a fall on the nates: the pustules were thinly scattered everywhere, except in the seat of former injury, and on this they were crowded as thickly as possible. Thus, too, when a part has been injured, and it may be, is healing, a disease having begun in the blood will manifest itself in this part. Impetigo appears about blows and scratches in unhealthy children; erysipelas about the same in men with unhealthy blood. Such facts as these appear to be sufficient evidences that morbid conditions of the blood are most probably the causes of the great majority of so-called spontaneous local inflammations; of such as cannot be traced to the direct influence of any external force, but appear, rather, as having an internal origin.

It may be added, that the state of the blood may determine not only the locality, but also the degree, and the form, and probable termination of the inflammation. But on this I need not dwell, having devoted a large portion of a former lecture to demonstrate that the products of even the same form of inflammation excited in the same tissue, may be different in different persons, according to the several peculiarities of their blood.

3. Respecting the disturbance of the third condition of healthy nutrition, namely, the due influence of the nervous force, as a constituent of the phenomena of inflammation, I have already spoken in a former lecture.* I now resume the subject, in the hope of showing that the disturbance of this condition may be one of the causes of inflammation.

To test the influence of the nervous force in engendering the inflammatory process, we must not, as is commonly done, take cases of the effects of external injury. Such an injury, or the presence of a foreign body, is supposed to excite inflammation by stimulating the nerves of the part, and by changing, through their influence, the state or action of the blood-vessels. This may be true; but we should remember that when a common injury is inflicted, it acts not only on the nerves of the part, but also on its proper tissues; and it may so affect the state of these tissues, that the changes produced in them may be the excitant of inflammation, independent of the affection of the nerves. All such cases as these are, thus, ambiguous. For a better test, we must select cases in which the excitant of inflammation acts (at least in the first instance) on the nervous system alone. Such cases are those already referred to. When the conjunctiva is inflamed after overworking of the eye, we cannot suppose that the light, by its direct contact, has affected the vessels, or the nutritive act, in the conjunctiva: it can, probably, affect either of these only through an influence reflected from the retina. So, when irritation of the urethra excites inflammation in the testicle; when the irritation of teething excites it in any distant part; when, as in a case quoted from Lallemand, by Dr. Williams, inflammation of the brain followed the application of a ligature to part of the brachial plexus;

* Lecture I. p. 12.

in these and the like cases—such as I mentioned in the first lecture,—we cannot but refer to the disturbance of the nervous force as the initiator of the phenomena of inflammation.

Now, for the explanation of such cases as these, there appear to be two chief theories—1. It may be that the nerves distributed to the minute blood-vessels of a part may be so affected that these vessels may dilate, and their dilatation may produce the other phenomena of inflammation; or, 2. The disturbance of the nervous force may more directly interfere with the process of nutrition, inasmuch as this force exercises always some influence in the nutrition of each part, and is (as one may say) one among the plasturgic forces.

The first of these theories has lately acquired a dominant place in systems of pathology, especially in those of Germany. The principal form of it, which has been maintained most prominently by Henle, has enlisted the approval of even Rokitansky, and is largely received, professing to explain all inflammations, and passing by the name of "neuro-pathological," to distinguish it from the "humoral" and all other theories of inflammation. This theory may be thus briefly stated. The exciting cause of inflammation, whether an external cause, such as an injury of a part, or an internal one, such as diseased blood, acts, in the first instance, on the sensitive centripetal or afferent nerves of the part. These it affects as a stimulant, producing in them an excited state, which state, being conveyed to some nervous centre, is thence reflected on the centrifugal or motor nerves of the blood-vessels of the same, or some other related, part. This reflection, however, is supposed to bring about a kind of antagonistic sympathy, such that, instead of exciting the motor forces of the blood-vessels to make them contract, it paralyses them, and is followed by their dilatation or relaxation. This dilatation being established, the exudation, and other phenomena of inflammation, are assumed to follow as natural, and most of them as mechanical, consequences.

The eminence of those who have supported this hypothesis makes one hesitate in rejecting it; and yet I cannot help believing it to be groundless. If we remember that parts may present some of the phenomena of inflammation, though they have no nerves, as the firmest tendons and articular cartilages; that the degrees of inflammation in parts bear no proportion to the amounts of pain in them when inflamed; that the severest pains may endure for very long periods with only trivial, if any, phenomena of inflammation; that the phenomena of the so-called reflex paralysis are rare, equivocal, and altogether insufficient for the foundation of a law or general principle; we may well think that there can be no sufficient ground for the invention of such an hypothesis as this. And, if we add that, even admitting the dilatation of bloodvessels as a possible consequence of the stimulus of sensitive nerves, yet the phenomena of even simple inflammation would be no necessary consequences thereof; that the varieties of inflammations would be quite unintelligible as results of similar mechanical disturbances of the circulation; and that the dilatation of bloodvessels, in any mechanical way produced, is followed by only feeble imitations of a part of the inflammatory process; then we may think that the hypothesis, if all its postulates be granted, will yet be insufficient for the explanation of the facts.

I believe that, if we would have any clear thoughts respecting the influence of the nerves in initiating inflammations, we must first receive the theory, referred to in the first lecture, that a certain exercise of the nervous force is habitually and directly engaged in the act of normal nutrition. If we admit this, there can be little difficulty in believing that the perturbations of the nervous force may engender the inflammatory mode of nutrition; especially when we see the normal and inflammatory modes connected through such intermediate instances as the increase of secretions when the nerves of a distant part are stimulated.

Now, that the nervous force has some other influence in normal nutrition than can be explained by referring to it only the government of the size of bloodvessels, we have, I think, ample evidence; and I cannot but wonder at the stedfastness with which some maintain or imply that the nervous force can manifest itself in nothing but impressions on the mind, and muscular contraction-force. So limited a view of the convertibility of nervous force, is such an one as the older electricians would have held, had they maintained that the only possible manifestations of electricity were the attractions and repulsions of light bodies, or that the electric force could never be made to appear in the form of magnetism, of chemical action, or of heat. We are too much shackled with these narrow dogmas of negation. The evidence of the correlation and mutual convertibility of the physical forces might lead us to anticipate a like variety of modes of manifestation for the nervous and other forces exercised in the living body.* We might

* For the suggestion of this view I have to thank my esteemed friend Dr. Carpenter, by whom I am glad to know that the subject of the correlation of the vital forces will shortly be fully illustrated.

anticipate, too, that, as the nervous force has its origin in the acts of nutrition by which the nerve-substance is formed, so, by reciprocal action, its exercise might affect the nutritive acts. As (for illustration sake) the completed blood affects all the processes by which itself was formed, so, we might suppose, would the nervous force be able to affect all the acts of which itself is the highest product.

But we need not be content with these suppositions of the direct influence of the nervous force on the nutritive act, while we remember such facts as these. The mind can affect all nutrition, but it can do so only by affecting, in the first instance, the nervous force; and its effects are such changes of nutrition as cannot be referred to mere changes of the size of the blood-vessels. Abstraction of the nervous influence from a part by division, or other profound injury, of its nerves, is, as a general rule, followed by serious impairment of its nutrition, and by such and so various impairments as cannot be accounted for by paralysis of the bloodvessels. The direct influcnee of the nervous system on both the quantity and the quality of secretions is a fact even commonly noticeable; and secretion and nutrition are so similar proccsses that we may be sure they are accomplished by the same forces similarly exercised.*

We seem, then, to have sufficient evidence that the nervous force is one of those which (at least in the highest animals) are engeged in the normal act of nutrition; and sufficient evidence that inflammation may ensue in consequence of disturbance of the nervous force, even when that force cannot have acted in the first instance on the bloodvessels of the inflamed part. I think, therefore, the expression is justified, that the inflammation of a part may have its origin in disturbance of the nervous force which is normally exercised in the nutrition of the part, and which is exercised directly, not merely through the government of the contractile coats of the blood-vessels.

The last of the necessary conditions of normal nutrition in a part is the healthy condition of the part itself. If a part be at present diseased, it will be apt to remain so, because of the continuance of the same diseased method of nutrition. Now, it appears highly probable that a disturbance of the healthy state of a part may introduce the phenomena of inflammation. This is probable for many reasons; as, first, from analogy with normal nutrition. Generally, the principal conditions of nutrition are the relative and mutual influences of the elements of the tissues and the blood. More particularly, the condition of the tissues determines, at least in great measure, both the quantity and the rate of movement of the blood supplied to them, the changes of the tissues, whether in growth or decrease, just preceding the adapted changes in the supply of blood. So we may believe a change in a part anyhow engendered may, by altering its relation to the blood, alter its mode of nutrition; and some of the changes may produce the inflammatory mode of nutrition, together with the altered supply of blood, and other characteristic signs.

Secondly, we may judge the same from the analogy between inflammation and the process of repair. Certainly it is the state of the injured part — *i. e.* of its proper tissues, not of its nerves and blood-vessels—which determines the process of repair: and some of the processes of repair are so like those of inflammation, that they are commonly identified, and are, perhaps, not capable of even a refined distinction.

And thirdly, the influence of the condition of the proper tissues of a part in initiating inflammation in it, is illustrated by more direct facts; such as, that injuries of parts that have no vessels or nerves are followed by altered modes of nutrition in them, these modes being more or less exact resemblances of inflammation. Thus, *e. g.* it is in the cornea, lens, vitreous humour, and the like, after injury.

On the whole, I think we may conclude that inflammation may have its origin in disturbance of the normal condition of the proper tissues of a part,—in such a disturbance as may be produced by injury, or by the proximity of disease. To this source, indeed, I should be disposed to refer nearly all inflammations that originate in the direct application of local stimuli, whether mechanical or chemical. It is true, that, in most cases, the stimulus affects at once the proper elements of the part, its nerves, and its bloodvessels, so that we cannot say how much of the disease is to be ascribed to the affection of each; but the fact that a process resembling, so far as it goes, that of inflammation may ensue after injury in parts that have neither vessels nor nerves, may make one believe that, in parts that have both, the inflammation depends mainly on injury, or other affection, of the proper tissue.

I have thus endeavoured to show that inflammation may take its rise, may have its proximate cause, in a disturbance of

* The subject is more fully illustrated in the Lectures on Nutrition, in the MEDICAL GAZETTE for 1847.

any one of the conditions of nutrition. In the examination of different cases, we find that, even while any three of the four chief conditions may be normal, yet a qualitative error of the fourth may bring in the phenomena of the inflammatory process and method of nutrition. In the necessity of choosing pointed cases, I may seem to have implied that it is usual for inflammation not only to begin, but to be maintained, by an error in one of the conditions of nutrition: but this is improbable. Rather we may believe, that many of the excitants of inflammation may affect at once more than one of the conditions of nutrition; and, as I stated in the first lecture, it is nearly certain that in every inflammation, after a short continuance, all the conditions of the nutritive process are alike involved in error.

P.S.—I cannot but fear, lest, having seldom referred, in these Lectures, to the works of those who have written on Inflammation before me, I may seem unready to acknowledge my obligations to them, or desirous to receive unmerited praise for originality of observation or of thought. Nothing is further from my intention, or, I believe, from my custom. The truth is, when I began to prepare for the delivery of the lectures, it seemed impossible to study their subject-matter by both reading and personal investigation: its extent seemed almost equally boundless in books and in nature. I therefore determined to occupy my time almost exclusively with personal inquiries and reflections; and to add the results of these to whatever I found true in the knowledge previously acquired by reading or by earlier examinations. If I had had time to study the vast literature of inflammation, I would gladly have endeavoured to assign to its proper author whatever fact or opinion of any value these lectures may contain; but this was impossible: and now, though conscious of being largely indebted, I am quite unable to say to what creditor each debt is due. But I gratefully acknowledge that my greatest obligations were incurred, many years ago, in reading the works on Inflammation of Hunter, Lawrence, Tweedie, James, and Macartney; and that, more recently, I have borrowed both facts and suggestions from the writings of Mr. Travers, Dr. Carpenter, Dr. C. J. B. Williams, and my valued friend and former pupil Mr. Humphry. Nor are my obligations less to Rokitansky, Henle, Virchow, Reinhardt, and, it may be, to many others from whom I may have derived such knowledge that I ought never to have forgotten whence it came.

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